Southern Medical Journal

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SEPTEMBER 1960

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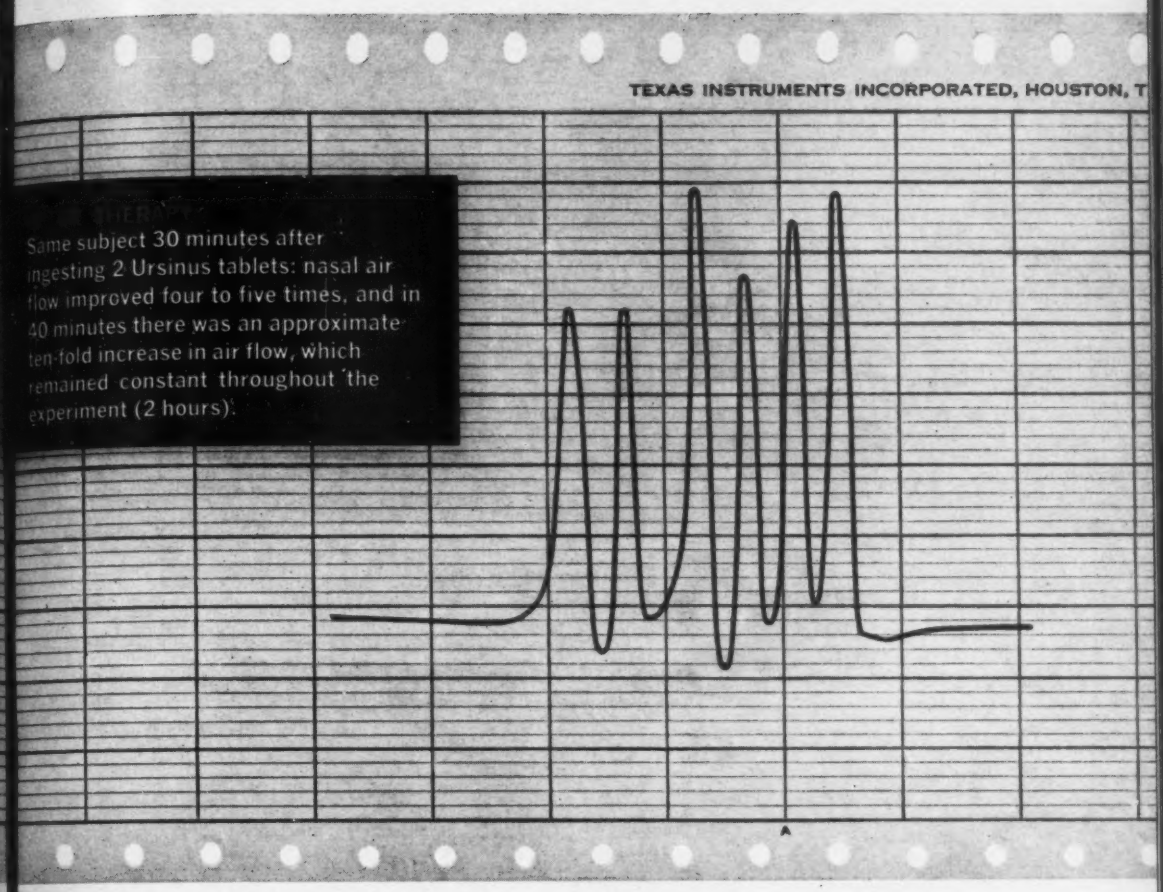
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References: 1. Steigmann, F., **et al.**: Am. J. Gastroenterol. 33:109 (Jan.) 1960. 2. Hock, C. W.: to be published. 3. Leming, B. H., Jr.: Clin. Med. 6:423 (Mar.) 1959. 4. Data in Roerig Medical Department Files.

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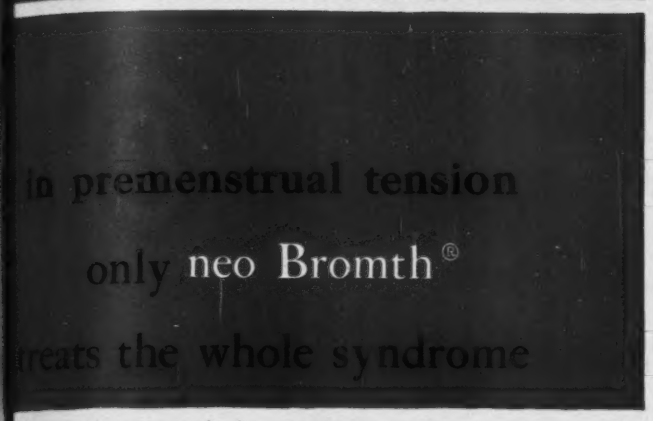

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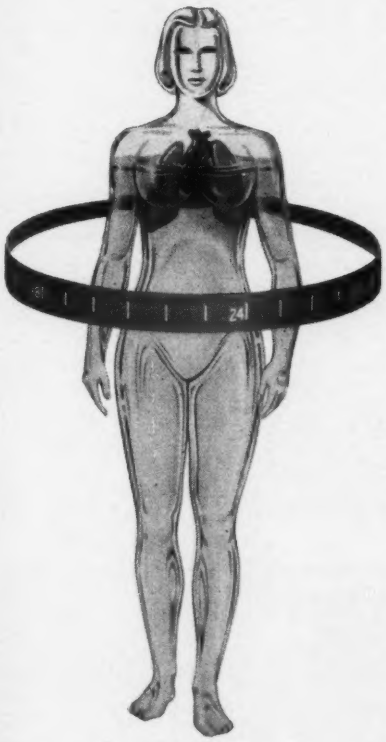

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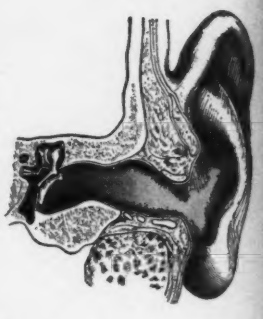

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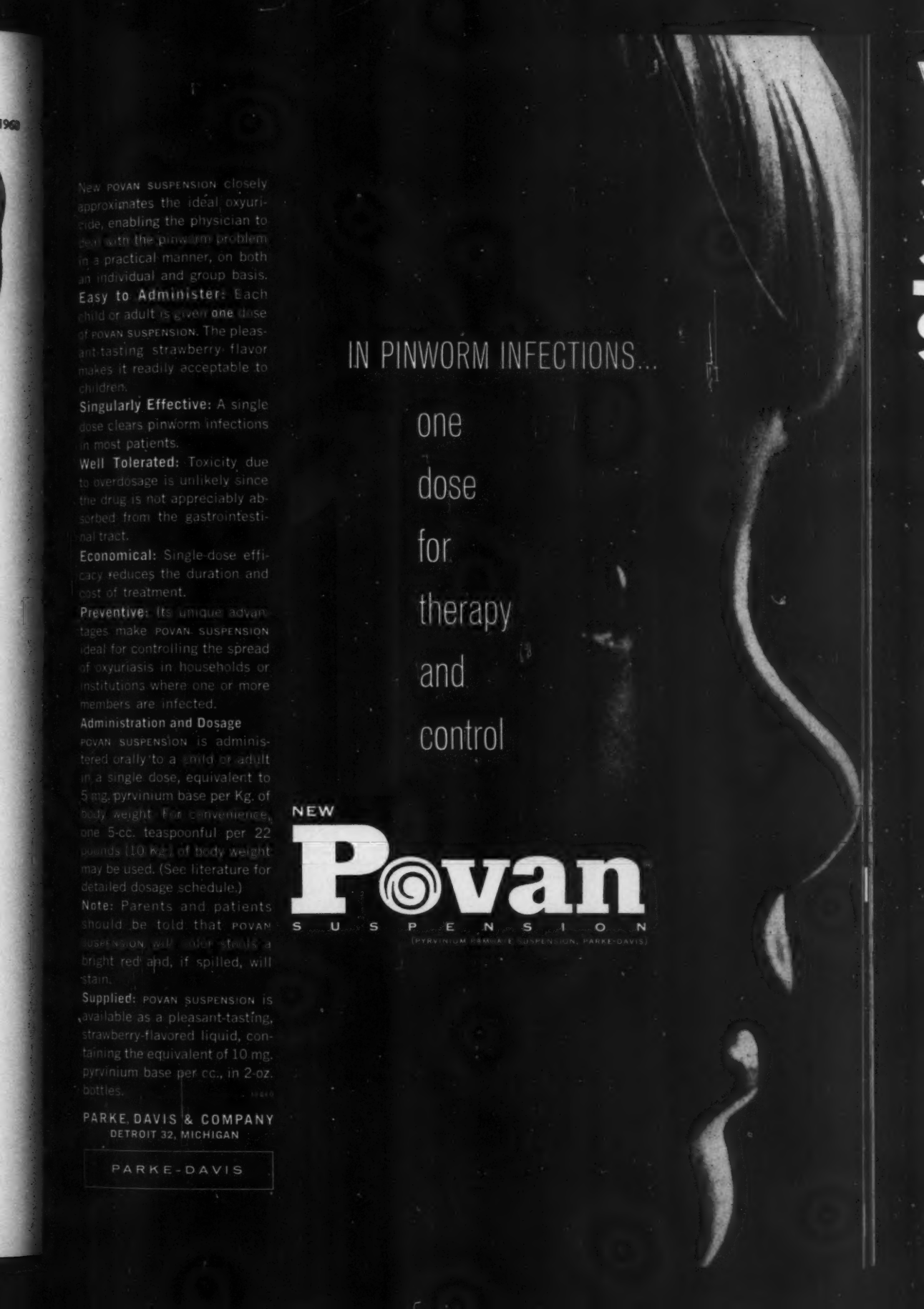

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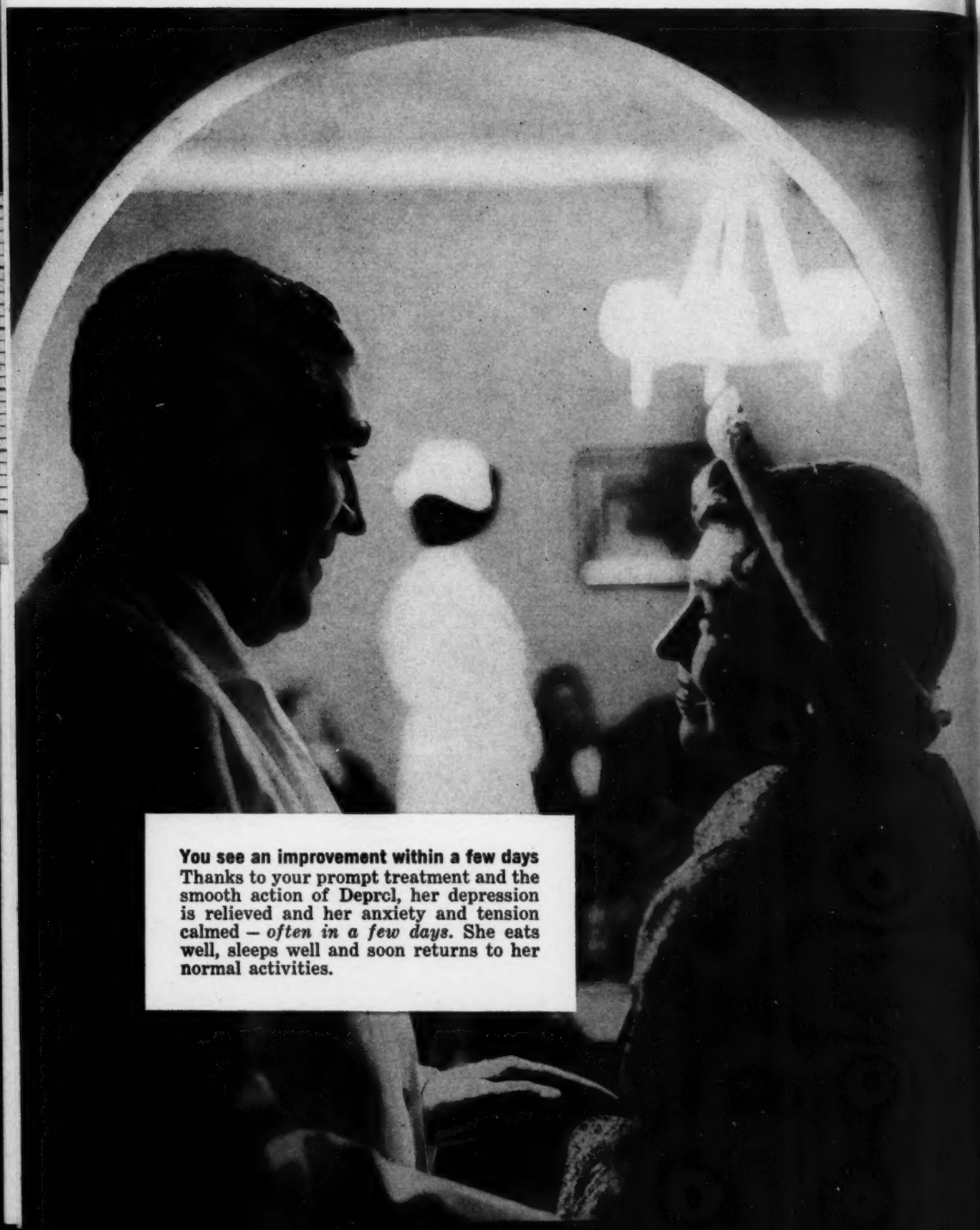

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Bibliography *(13 clinical studies, 858 patients):* **1.** Alexander, L. (35 patients): Chemotherapy of depression – Use of meprobamate combined with benactyzine (2-diethylaminoethyl benzilate) hydrochloride. J.A.M.A. 166:1019, March 1, 1958. **2.** Bateman, J. C. and Carlton, H. N. (50 patients): Meprobamate and benactyzine hydrochloride (Deprol) as adjunctive therapy for patients with advanced cancer. Antibiotic Med. & Clin. Therapy 6:648, Nov. 1959. **3.** Beerman, H. M. (44 patients): The treatment of depression with meprobamate and benactyzine hydrochloride. Western Med. 1:10, March 1960. **4.** Bell, J. L., Tauber, H., Santy, A. and Pulito, F. (77 patients): Treatment of depressive states in office practice. Dis. Nerv. System 20:263, June 1959. **5.** Breitner, C. (31 patients): On mental depressions. Dis. Nerv. System 20:142, (Section Two), May 1959. **6.** Gordon, P. E. (50 patients): Deprol in the treatment of depression. Dis. Nerv. System 21:215, April 1960. **7.** Landman, M. E. (50 patients): Clinical trial of a new antidepressive agent. J. M. Soc. New Jersey. In press, 1960. **8.** McClure, C. W., Papas, P. N., Speare, G. S., Palmer, E., Slattery, J. J., Konefal, S. H., Henken, B. S., Wood, C. A. and Ceresia, G. B. (128 patients): Treatment of depression – New technics and therapy. Am. Pract. & Digest Treat. 10:1525, Sept. 1959. **9.** Pennington, V. M. (135 patients): Meprobamate-benactyzine (Deprol) in the treatment of chronic brain syndrome, schizophrenia and senility. J. Am. Geriatrics Soc. 7:656, Aug. 1959. **10.** Rickels, K. and Ewing, J. H. (35 patients): Deprol in depressive conditions. Dis. Nerv. System 20:364, (Section One), Aug. 1959. **11.** Ruchwarger, A. (87 patients): Use of Deprol (meprobamate combined with benactyzine hydrochloride) in the office treatment of depression. M. Ann. District of Columbia 28:438, Aug. 1959. **12.** Settel, E. (52 patients): Treatment of depression in the elderly with a meprobamate-benactyzine hydrochloride combination. Antibiotic Med. & Clin. Therapy 7:28, Jan. 1960. **13.** Splitter, S. R. (84 patients): Treatment of the anxious patient in general practice. J. Clin. & Exper. Psychopath. In press, April-June 1960.

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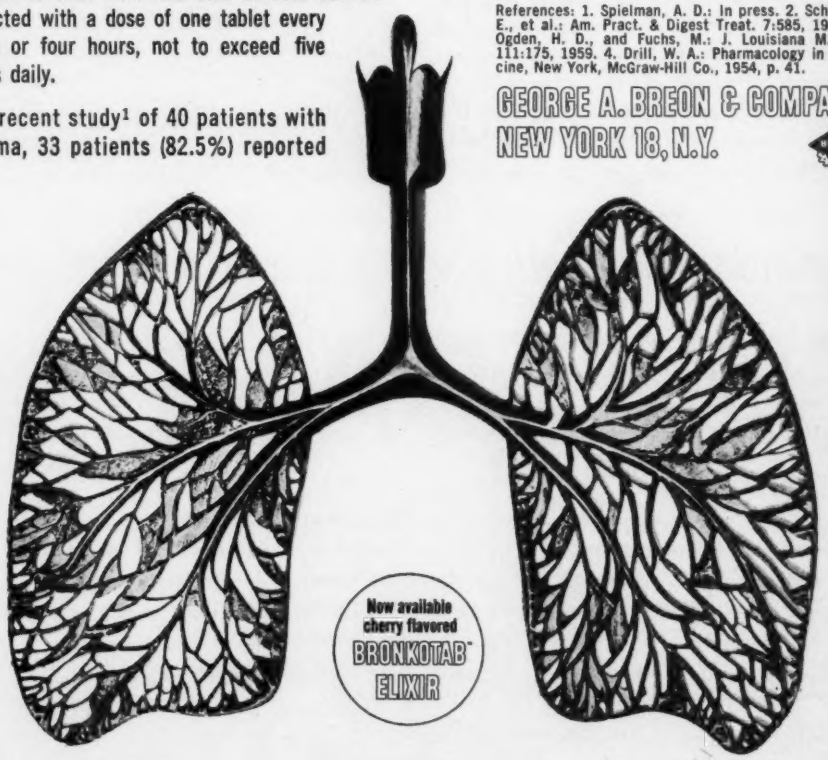

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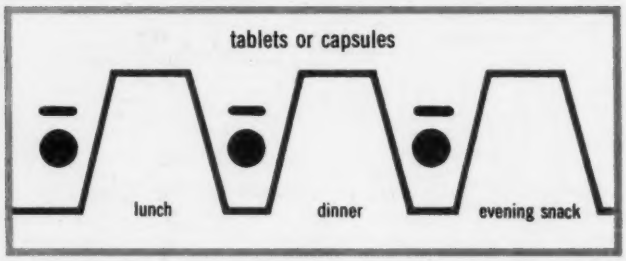
9,000 TONS
OF EXCESS WEIGHT
LOST
—OBESITY CHART—
WEIGHT
Case histories show that patients lose an average of 6 lbs. per 100 tablets of Obedrin—making a total weight loss of 18,000,000 lbs. since Obedrin was introduced.
Obedrin and the 60-10-70 Basic Plan succeed where other reducing regimens fail. The Obedrin Basic Plan provides three essentials for weight reduction:
1. Supervision by the physician—Patients need the emotional support and discipline that only the physician can contribute.
2. A balanced eating plan—The 60-10-70 Basic Plan offers the patient a wide variety of menus, carefully calculated to supply a minimum of 60 grams of carbohydrate, 10 grams of fat, and 70 grams of protein each day, with *no* calory counting.
3. Supportive medication—Each capsule or tablet of Obedrin provides:
• Semoxydrine® HCl (methamphetamine HCl), 5 mg., for its anorexigenic and mood-lifting effects
• Pentobarbital, 20 mg., to guard against excitation
• Thiamine Mononitrate, 0.5 mg., Riboflavin, 1 mg., and Nicotinic Acid (Niacin), 5 mg., to supplement the diet
• Ascorbic Acid, 100 mg., to help mobilize tissue fluids
Obedrin and the 60-10-70 Basic Plan help the overweight patient establish correct eating habits....first, to lose excess pounds and then, more important, to *maintain* optimum weight.
Obedrin®
and the 60-10-70 Basic Plan
provide an effective weight-control regimen
THE R. E. MASSENGILL COMPANY
Bristol, Tennessee
New York • Kansas City • San Francisco

A FLEXIBLE DOSAGE FORM PROVIDES DEPENDABLE CONTROL OF APPETITE



The Obedrin formula permits a flexible dosage schedule to depress the appetite at peak hunger periods. The physician can adjust dosage to fit each patient's need.

ADVANTAGES OF OBEDRIN—

A dependable anorexigenic agent
A flexible dosage form
Minimal central nervous stimulation
Vitamins to supplement the diet

Supplied: In bottles of 100, 500, and 1,000

Write for 60-10-70 Basic Plan menus and weight charts.

Obedrin®

and the 60-10-70 Basic Plan

THE S. E. MASSENGILL COMPANY Bristol, Tennessee • New York • Kansas City • San Francisco

"...A SIGNIFICANT MAJOR ADVANCE IN THE MANAGEMENT OF TINEA CAPITIS."*

GRIFULVIN®

Griseofulvin

FIRST ORALLY EFFECTIVE AGENT IN RINGWORM

WELL TOLERATED • OBVIATES NEED FOR X-RAY EPILATION • USUALLY CLEARS SCALP RINGWORM WITHIN 4 TO 6 WEEKS

Dosage: Adults—250 mg. q.i.d. or 500 mg. b.i.d. Children—According to weight, 250 mg. to 1.0 Gm. daily, in divided doses.

Supplied: new 500 mg. scored yellow tablets, bottles of 20 and 100; and 250 mg. scored aquamarine tablets, bottles of 16 and 100.

*Newcomer, V. D., **et al.**: A.M.A. J. Dis. Child. **99**:585, 1960.

McNEIL LABORATORIES, INC • PHILADELPHIA 32, PA.

McNEIL

Tinea capitis, before GRIFULVIN. Microscopic and Wood's light examinations help differentiate this infection from non-ringworm disorders.

After 5 weeks' treatment with GRIFULVIN. (Photo taken 1½ months after discontinuance of medication.)

Fostex® treats their acne while they wash

completely emulsifies and washes off excess oil from the skin.

penetrates and softens comedones, unblocks pores and facilitates removal of sebum plugs.

removes papule coverings and permits drainage of sebaceous glands.

Patients like Fostex because it is so easy to use. They simply wash acne skin 2 to 4 times a day with Fostex Cream or Fostex Cake, instead of using soap.

Fostex contains Sebulytic®,* a combination of surface-active wetting agents with remarkable antiseborrheic, keratolytic and antibacterial actions . . . enhanced by sulfur 2%, salicylic acid 2%, and hexachlorophene 1%.

*sodium lauryl sulfoacetate, sodium alkyl aryl polyether sulfonate and sodium dioctyl sulfosuccinate.

Fostex is available in two forms—

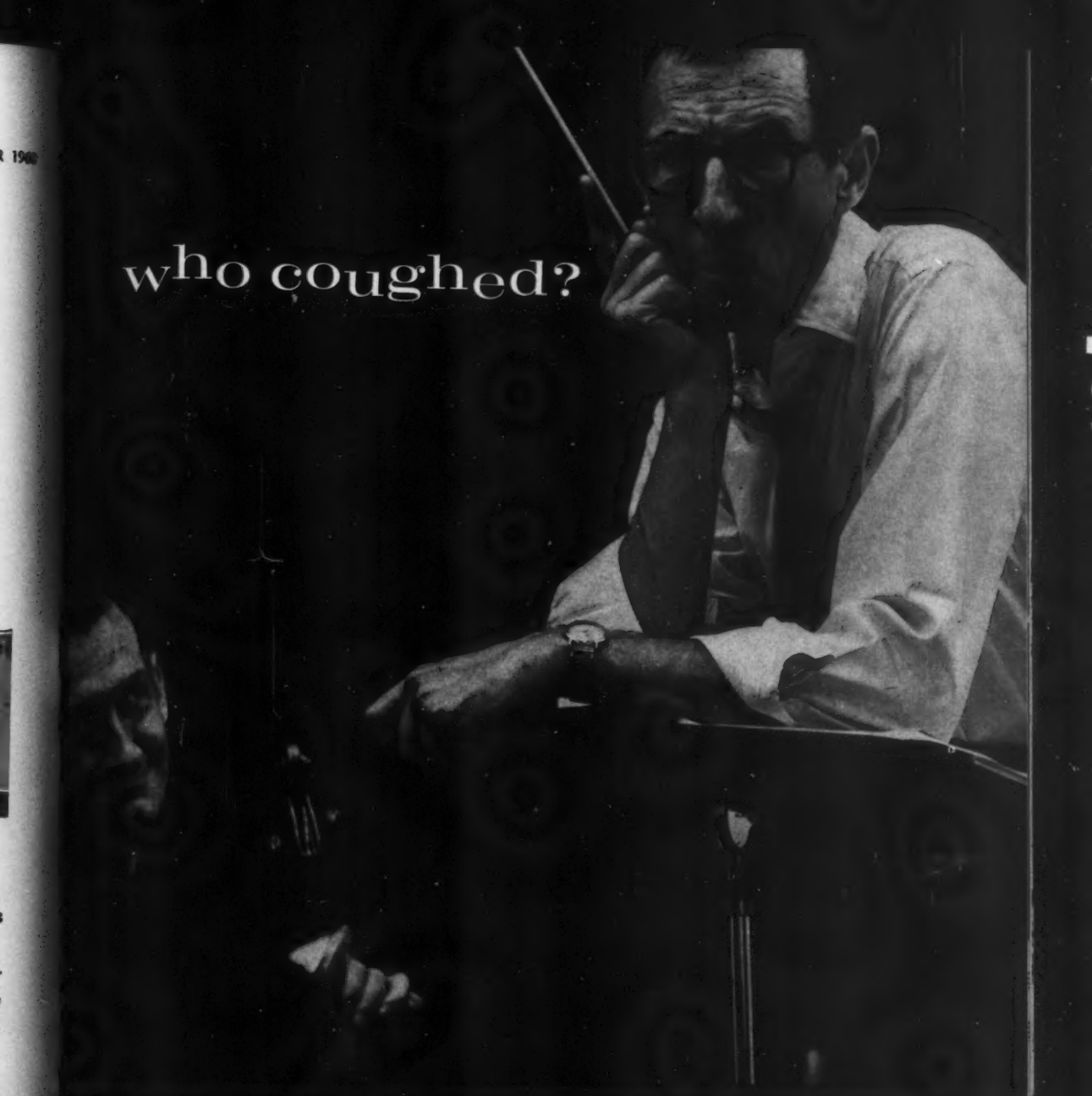
FOSTEX CREAM, in 4.5 oz. jars.
FOSTEX CAKE, in bar form.

Fostex Cream and Fostex Cake are interchangeable for therapeutic washing of the skin. Fostex Cream is approximately twice as drying as Fostex Cake.

Fostex Cream is also used as a therapeutic shampoo in dandruff and oily scalp.

Write for samples.

WESTWOOD PHARMACEUTICALS • Buffalo 13, New York



WHENEVER COUGH THERAPY IS INDICATED

THE COMPLETE Rx FOR COUGH CONTROL

Syrup

cough | sedative | antihistamine
decongestant | expectorant

▪ relieves cough and associated symptoms in 15-20 minutes ▪ effective for 6 hours or longer ▪ promotes expectoration ▪ rarely constipates ▪ agreeably cherry-flavored

Each teaspoonful (5 cc.) of HYCOMINE® Syrup contains:

Hycodan®		
Dihydrocodeinone Bitartrate (Warning: May be habit-forming)	5 mg.	6.5 mg.
Homatropine Methylbromide	1.5 mg.	
Pyrilamine Maleate		12.5 mg.
Phenylephrine Hydrochloride		10 mg.
Ammonium Chloride		60 mg.
Sodium Citrate		85 mg.

Average adult dose: One teaspoonful after meals and at bedtime. May be habit-forming. Federal law permits oral prescription.

Literature on request

ENDO LABORATORIES
Richmond Hill 18, New York

In over five years

...for the tense and nervous patient

Despite the introduction in recent years of "new and different" tranquilizers, Miltown continues, quietly and steadfastly, to gain in acceptance. Meprobamate (Miltown) is prescribed by the medical profession more than any other tranquilizer in the world.

The reasons are not hard to find. Miltown is a ***known*** drug. Its few side effects have been fully reported. ***There are no surprises in store for either the patient or the physician.***

# of clinical use...

Proven

in more than 750 published clinical studies

Effective

for relief of anxiety and tension

Outstandingly Safe

1 simple dosage schedule produces rapid, reliable tranquilization without unpredictable excitation

2 no cumulative effects, thus no need for difficult dosage readjustments

3 does not produce ataxia, change in appetite or libido

4 does not produce depression, Parkinson-like symptoms, jaundice or agranulocytosis

5 does not impair mental efficiency or normal behavior

Miltown®

meprobamate (Wallace)

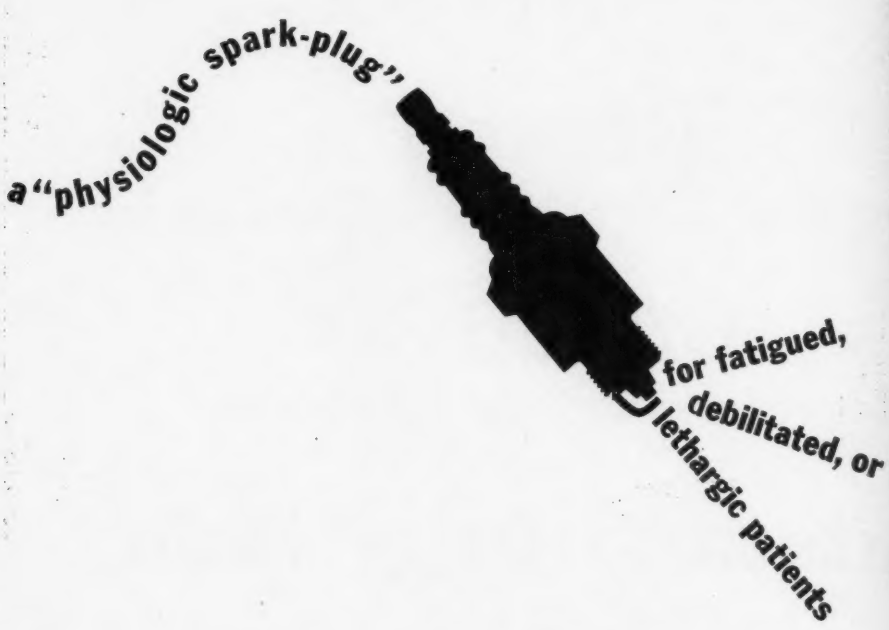
Usual dosage: One or two 400 mg. tablets t.i.d.
Supplied: 400 mg. scored tablets, 200 mg. sugar-coated tablets; or as MEPROTABS®—400 mg. *unmarked*, coated tablets.

WALLACE LABORATORIES / *Cranbury, N. J.*

CM-2333

Nico-Metrazol™

the safe ergogenic agent



Whenever non-specific fatigue indicates the need for safe cerebral stimulation, **Nico-Metrazol** deserves a therapeutic trial. The gentle activation of the central nervous system and the increased rate of **cerebral** blood flow effected by Metrazol®, combined with increased **peripheral** blood flow induced by nicotinic acid, provide a "physiologic spark-plug" for renewed mental and physical vitality. Fatigue and listlessness are frequently replaced by a resurgence of energy and alertness. Chronically apathetic geriatric patients often show significant improvement on Nico-Metrazol therapy.

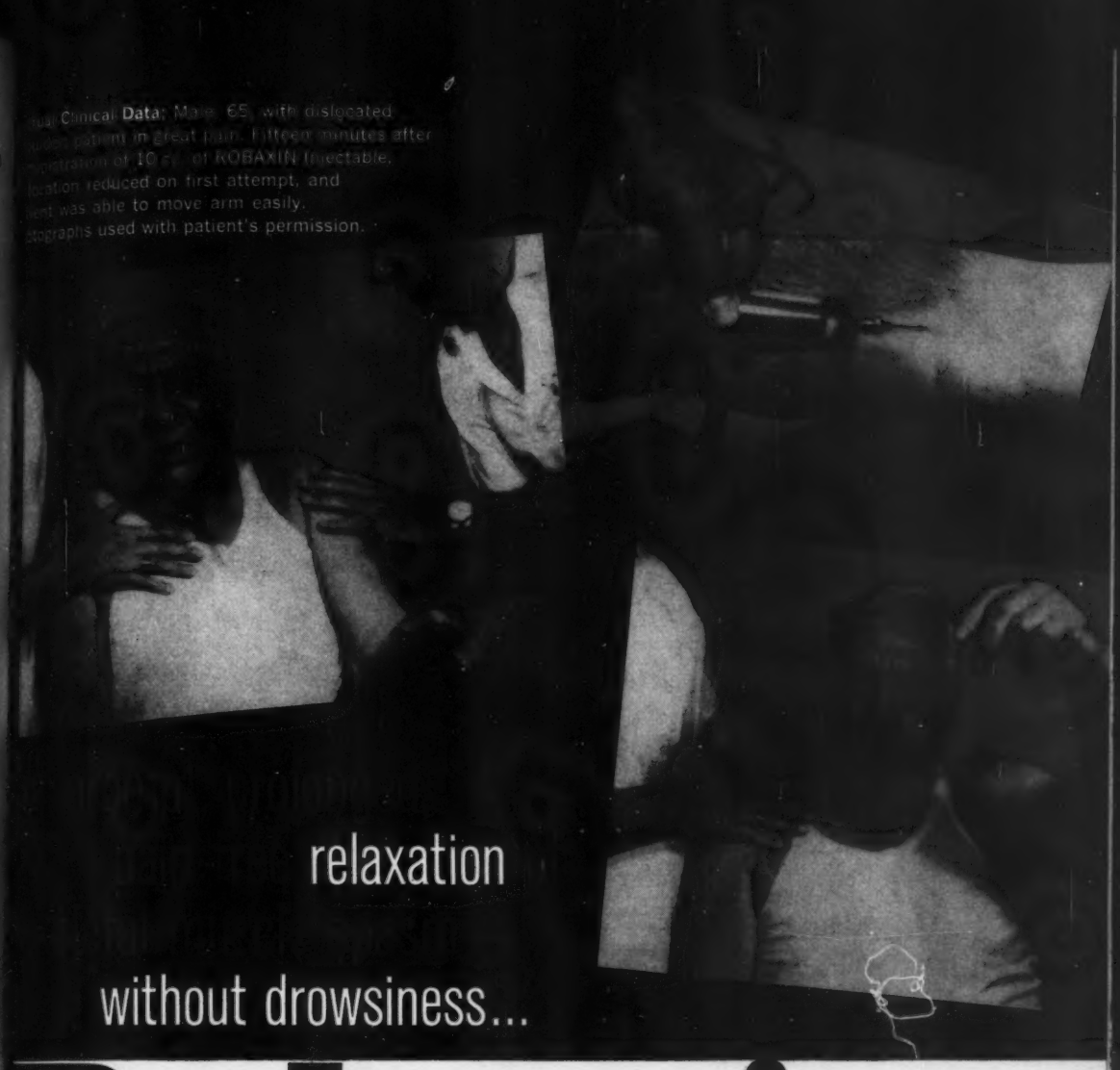
Outstanding Safety: Nico-Metrazol is markedly free of undesirable systemic action. It causes neither hypertension nor postural hypotension. Nico-Metrazol has no adverse effect on liver or bone marrow functions. The vasodilating action of nicotinic acid produces a transient flush and is accompanied by a feeling of warmth and stimulation.

Dosage: **Initially,** 2 Nico-Metrazol Tablets, or 2 teaspoonfuls of Nico-Metrazol Elixir, three or four times daily. **For maintenance,** after optimal results have been achieved, dosage may be reduced to 1 Nico-Metrazol Tablet or 1 teaspoonful of Nico-Metrazol Elixir three times daily.

Supply: Nico-Metrazol Tablets and Elixir—100 mg. Metrazol and 50 mg. nicotinic acid in each tablet or teaspoonful.

For information on Vita-Metrazol and Metrazol dosage forms, consult your current Physicians' Desk Reference.

KNOLL PHARMACEUTICAL COMPANY • ORANGE, NEW JERSEY


Clinical Data: Male, 65, with dislocated
patient in great pain. Fifteen minutes after
of 10 cc. of ROBAXIN Injectable,
reduced on first attempt, and
was able to move arm easily.
graphs used with patient's permission.

relaxation

without drowsiness...

Robaxin®

Robins

methocarbamol 'Robins' U.S. Pat. No. 2770649

ROBAXIN Injectable: for relaxation of painful spasm within minutes.

ROBAXIN Tablets: for initial relief, or to maintain relaxation originally induced by ROBAXI Injectable. Virtually free from adverse side effects, including drowsines

Ten published studies show ROBAXIN Injectable and ROBAXIN Tablets beneficial in 91 of cases.[1-10] Literature available to physicians on request.

SUPPLY: ROBAXIN Tablets, 0.5 Gm. (white, scored) in bottles of 50 and 500.

ROBAXIN Injectable, each ampul containing 1.0 Gm. of methocarbamol in 10 cc. of steri solution.

A. H. ROBINS CO., INC., Richmond 20, Virginia

GOTISTOP ANTS VERTIVERT

ANTIGO VERTS STOPIANT

ANTISTOP VERTS GOTIVERT

ANTIVERT STOPS VERTIGO
(virtually 9 times out of 10)

Remission in 82%; relief in 92%. So reports an investigator who recently studied ANTIVERT in dizziness.[1] After studying 50 patients, Scal concluded that "Those with Meniere's syndrome who were given the preparation [ANTIVERT] in the early stages of this condition, reported prompt improvement in the relief of dizziness, headaches and tinnitus."[1]

ANTIVERT combines meclizine (12.5 mg.) with nicotinic acid (50 mg.). Prescribe one ANTIVERT tablet before each meal for relief of Meniere's syndrome, arteriosclerotic vertigo, labyrinthitis, and vertigo of nonspecific origin.

Supplied: In bottles of 100 blue-and-white scored tablets. Prescription only.

Reference: 1. Scal, J. C.: Eye Ear Nose & Throat Month. 38:738 (Sept.) 1959.

Antivert®

ROERIG

New York 17, N. Y.
Division, Chas. Pfizer & Co., Inc.
Science for the World's Well-Being™

and to help combat the nutritional problems of aging... NEOBON® capsules five-factor geriatric supplement

ANTIQUE

suturing: Xylocaine® HCl Solution applied topically will permit cleaning and suturing of wounds with patient comfort in an emergency or in the office. Fast acting – Safe – Dependable.

bursitis: Xylocaine HCl Solution injected into the painful area will diffuse around the bursae relieving pain promptly – often restoring normal freedom of motion. Prolonged anesthesia often prevents recurring pain.

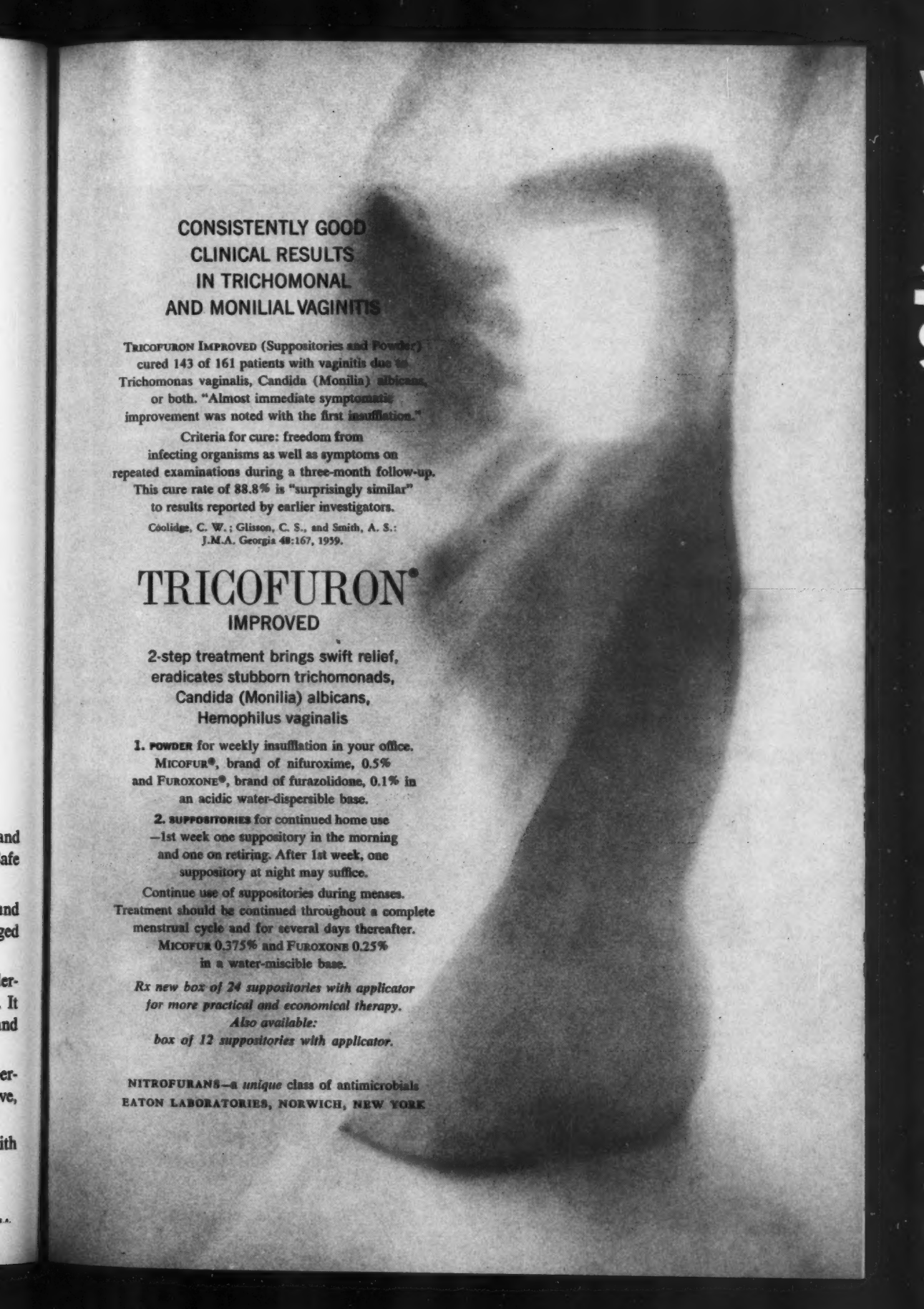
therapeutic block: Xylocaine HCl Solution interrupts the underlying mechanism of pain, with relief often persisting even after the block has disappeared. It is of value in assisting motion or manipulation; for severe, intractable pain conditions; and in allowing patient comfort for other procedures.

minor surgery: Xylocaine HCl Solution will diffuse over a wide operative field, permitting pain-free removal of warts, cysts, moles, etc., and giving safe, effective, and predictable anesthesia for patient comfort.

Supplied: Multiple dose vials, 20 cc. and 50 cc.; 0.5%, 1% and 2% without and with epinephrine 1:100,000. Ampules, 2 cc.; 2% without and with epinephrine 1:100,000.

ASTRA

*U.S. PAT. NO. 2,441,498 MADE IN U.S.A.



CONSISTENTLY GOOD CLINICAL RESULTS IN TRICHOMONAL AND MONILIAL VAGINITIS

TRICOFURON IMPROVED (Suppositories and Powder) cured 143 of 161 patients with vaginitis due to Trichomonas vaginalis, Candida (Monilia) albicans, or both. "Almost immediate symptomatic improvement was noted with the first insufflation."

Criteria for cure: freedom from infecting organisms as well as symptoms on repeated examinations during a three-month follow-up. This cure rate of 88.8% is "surprisingly similar" to results reported by earlier investigators.

Coolidge, C. W.; Glisson, C. S., and Smith, A. S.: J.M.A. Georgia 48:167, 1959.

TRICOFURON®

IMPROVED

2-step treatment brings swift relief, eradicates stubborn trichomonads, Candida (Monilia) albicans, Hemophilus vaginalis

1. **POWDER** for weekly insufflation in your office. MICOFUR®, brand of nifuroxime, 0.5% and FUROXONE®, brand of furazolidone, 0.1% in an acidic water-dispersible base.

2. **SUPPOSITORIES** for continued home use —1st week one suppository in the morning and one on retiring. After 1st week, one suppository at night may suffice.

Continue use of suppositories during menses. Treatment should be continued throughout a complete menstrual cycle and for several days thereafter. MICOFUR 0.375% and FUROXONE 0.25% in a water-miscible base.

Rx new box of 24 suppositories with applicator for more practical and economical therapy.
Also available:
box of 12 suppositories with applicator.

NITROFURANS—a *unique* class of antimicrobials
EATON LABORATORIES, NORWICH, NEW YORK

a promise fulfilled

All corticosteroids provide symptomatic control in rheumatoid arthritis, inflammatory dermatoses, and bronchial asthma. They *differ* in the frequency and severity of side effects. Introduced in 1958, ARISTOCORT Triamcinolone bore the promise of high efficacy and relative safety. Physicians today recognize that the promise has been fulfilled . . . as evidenced by the high rate of refilled ARISTOCORT prescriptions.

Aristocort Triamcinolone LEDERLE

Lederle LEDERLE LABORATORIES, A Division of AMERICAN CYANAMID COMPANY, Pearl River, N.Y.

Diagnostic Quandaries

Colitis? Gall Bladder Disease?
Chronic Appendicitis?
Rheumatoid Arthritis? Regional Enteritis?

A DISEASE that is frequently overlooked in solving diagnostic quandaries is amebiasis. Its symptoms are varied and contradictory, and diagnosis is extremely difficult. In one study, 56% of the cases would have been overlooked if the routine three stool specimens had been relied on.[1]

Another study found 96% of a group of 150 patients with rheumatoid arthritis were infected by *E. histolytica*. In 15 of these subjects, nine stool specimens were required to establish the diagnosis.[2]

Webster discovered amebic infection in 147 cases with prior diagnoses of spastic colon, psychoneurosis, gall bladder disease, nervous indigestion, chronic appendicitis, and other diseases. Duration of symptoms varied from one week to over 30 years. In some cases, it took as many as six stool specimens to establish the diagnosis of amebiasis.[3]

Now treatment with Glarubin provides a means of differential diagnosis in suspected cases of amebiasis. Glarubin, a crystalline glycoside obtained from the fruit of *Simarouba glauca*, is a safe, effective amebicide. It contains no arsenic, bismuth, or iodine. Its virtual freedom from toxicity makes it practical to treat suspected cases without undertaking difficult, and frequently undependable, stool analyses. Marked improvement following administration of Glarubin indicates pathologically significant amebic infection.

Glarubin is administered orally in tablet form and does not require strict medical supervision or hospitalization. Extensive clinical trials prove it highly effective in intestinal amebiasis.

Glarubin*

TABLETS

specific for intestinal amebiasis

Supplied in bottles of 40 tablets, each tablet containing 50 mg. of glaucarubin.

Write for descriptive literature, bibliography, and dosage schedules.

1. Cook, J.E., Briggs, G.W., and Hindley, F.W.: Chronic Amebiasis and the Need for a Diagnostic Profile, Am. Pract. and Dig. of Treat. *6*:1821 (Dec., 1955).

2. Rinehart, R.E., and Marcus, H.: Incidence of Amebiasis in Healthy Individuals, Clinic Patients and Those with Rheumatoid Arthritis, Northwest Med., *54*:708 (July, 1955).

3. Webster, B.H.: Amebiasis, a Disease of Multiple Manifestations, Am. Pract. and Dig. of Treat. *9*:897 (June, 1958).

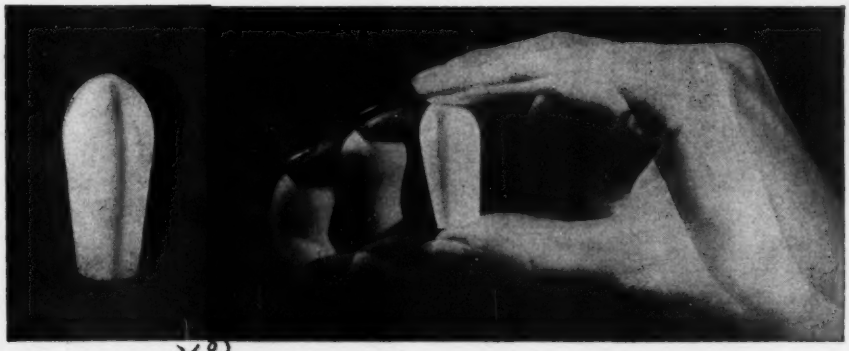
*U.S. Pat. No. 2,864,745

THE S.E. MASSENGILL COMPANY
BRISTOL, TENNESSEE
NEW YORK • KANSAS CITY • SAN FRANCISCO

Against the "Vaginitis Spectrum"

TRICHOMONAS
MONILIA
BACTERIA

A welcome clinical advance...
effective medication
in an appealing form



Soft and pliant as a tampon, the Milibis vaginal suppository offers proved therapeutic action* in a vehicle giving unusual clinical advantages to both patients and physician.

COVERS CERVIX AND VAGINAL WALL—The pliant Milibis suppository disintegrates readily and molds itself to the cervix as well as the columns and rugae of the vaginal vault.

SHORT DOSAGE SCHEDULE—The short course of treatment with Milibis—only 10 suppositories in most cases—together with the clean, odorless, non-staining qualities eliminates psychic barriers which often interrupt longer treatments before complete cure.

SUPPLIED: BOXES OF 10 with applicator.

Now supplied with plastic applicator
- SANITARY
- INSURES CORRECT SUPPOSITORY PLACEMENT

MILIBIS® Vaginal Suppositories

Winthrop LABORATORIES
New York 18, N. Y.

*97 per cent effective in a study of 564 cases; 94 per cent effective in a series of 510 cases.

Milibis (brand of glycobiarsol), trademark reg. U. S. Pat. Off.

Novahistine® works better than antihistamines alone

Stuffy, runny noses... swollen, weepy eyes are more effectively relieved with Novahistine. The distinctly additive action of the vasoconstrictor and antihistamine combined in Novahistine relieves allergic symptoms more effectively than either drug alone.

One dose of 2 tablets for day-long or night-long relief. Each long-acting tablet contains 25 mg. phenylephrine HCl and 4 mg. chlorprophenpyridamine maleate. Bottles of 50 and 250 green, film-coated tablets.

PITMAN-MOORE COMPANY Division of Allied Laboratories, Inc., Indianapolis 6, Indiana

Novahistine LP LONG-ACTING

Now—All cold symptoms can be controlled

Tussagesic

timed-release *tablets*

Controls congestion
with Triaminic,[1,2,3] the leading oral nasal decongestant.

Controls aches and fever
with well-tolerated APAP, non-addictive analgetic[4] and excellent antipyretic.[5]

Each TUSSAGESIC Tablet provides:

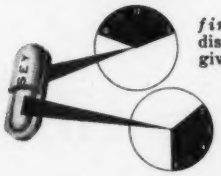
TRIAMINIC®	50 mg.
(phenylpropanolamine HCl	25 mg.
pheniramine maleate	12.5 mg.
pyrilamine maleate	12.5 mg.)
Dormethan (brand of dextromethorphan HBr)	30 mg.
Terpin hydrate	180 mg.
APAP (N-acetyl-p-aminophenol)	325 mg.

References: 1. Lhotka, F. M.: Illinois M. J. 112:259 (Dec.) 1957. 2. Fabricant, N. D.: E.E.N.T. Monthly 37:460 (July) 1958. 3. Farmer, D. F.: Clin. Med. 5:1183 (Sept.) 1958. 4. Bonica, J. J.: in Drugs of Choice, Mosby, St. Louis, 1958, p. 272. 5. Dascomb, H. E.: in Current Therapy, Saunders, Phila., 1958, p.78. 6. Bickerman, H. A.: in Drugs of Choice, Mosby, St. Louis, 1958, p.547.

Controls cough centrally
with non-narcotic Dormethan, possessing "amply demonstrated" antitussive activity,[6] as effective as codeine.

Liquefies tenacious mucus
with terpin hydrate, classic expectorant.

Prompt and prolonged relief because of this special "timed release" design:



first – the outer layer dissolves within minutes to give 3 to 4 hours of relief

then – the inner core releases its ingredients to sustain relief for 3 to 4 more hours

Dosage: One tablet in the morning, midafternoon and at bedtime. Pediatric dosage chart for Tussagesic Suspension available on request.

TUSSAGESIC SUSPENSION provides palatability and convenience which make it especially attractive to children and other patients who prefer liquid medication.

SMITH-DORSEY • a division of The Wander Company • Lincoln, Nebraska

CLINICAL REMISSION IN A "PROBLEM" ARTHRITIC

In rheumatoid arthritis with serious corticoid side effects. Following profound weight loss and acute g.i. distress on prednisolone, a 45-year-old bookkeeper with a five-year history of severe arthritis was started on DECADRON, 1 mg./day. Dosage was promptly reduced to 0.5 mg./day. After ten months on DECADRON, she gained back eleven pounds, feels very well, and had no recurrence of stomach symptoms. She is in clinical remission.*

Now convenient b.i.d. alternate dosage schedule: the degree and extent of relief provided by DECADRON allows for b.i.d. maintenance dosage in many patients with so-called "chronic" conditions. Acute manifestations should first be brought under control with a t.i.d. or q.i.d. schedule.

Supplied: As 0.75 mg. and 0.5 mg. scored, pentagon-shaped tablets in bottles of 100. Also available as injection DECADRON Phosphate. Additional information on DECADRON is available to physicians on request. DECADRON is a trademark of Merck & Co., Inc.

*From a clinical investigator's report to Merck Sharp & Dohme.

Decadron®

Dexamethasone

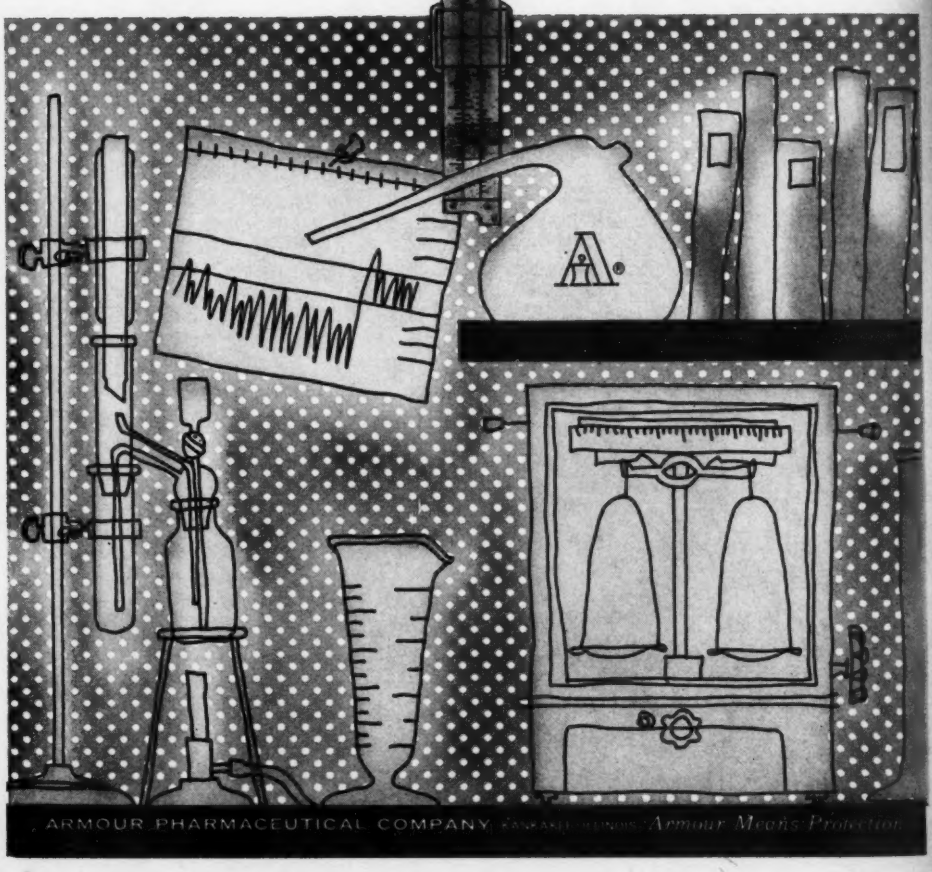
TREATS MORE PATIENTS MORE EFFECTIVELY

MERCK SHARP & DOHME · Division of Merck & Co., Inc., West Point, Pa.

ALWAYS SPECIFY ARMOUR THYROID

ARMOUR THYROID for over half a century has been more widely prescribed...more widely dispensed than any other thyroid product. Pioneer in thyroid standardization, Armour's rich background of experience assures you of unsurpassed quality, uniform potency and consistent therapeutic effects.

BY ANY MEASUREMENT THE THYROID OF CHOICE



the complaint: "nervous indigestion"

the diagnosis: any of several nonspecific and functional gastrointestinal disorders requiring relief of symptoms by sedative-antispasmodic action with concomitant digestive enzyme therapy.

the prescription: a new formulation incorporated in an enteric-coated tablet, providing the multiple actions of widely accepted Donnatal® and Entozyme.®

the dosage: two tablets three times a day, or as indicated.

in the gastric-soluble outer layer:

Hyoscyamine sulfate	0.0518 mg.
Atropine sulfate	0.0097 mg.
Hyoscine hydrobromide	0.0033 mg.
Phenobarbital (1/8 gr.)	8.1 mg.
Pepsin, N. F.	150 mg.

in the enteric-coated core:

Pancreatin, N. F.	300 mg.
Bile salts	150 mg.

antispasmodic • sedative • digestant

DONNAZYME®

A. H. ROBINS COMPANY, INCORPORATED • RICHMOND 20, VIRGINIA

PABALATE®

Robins

COMBINING MUTUALLY SYNERGISTIC NON-STEROID ANTIRHEUMATICS

"superior to aspirin" — ". . . evidence seems to indicate that the concurrent administration of para-aminobenzoic and salicylic acid [as in Pabalate] produces a more uniformly sustained level for prolonged analgesia and, therefore, is superior to aspirin in the treatment of chronic rheumatic disorders."[1]

In each yellow enteric-coated **PABALATE** tablet:

Sodium salicylate (5 gr.)	0.3 Gm.
Sodium para-aminobenzoate (5 gr.)	0.3 Gm.
Ascorbic acid	50.0 mg.

For the patient who should avoid sodium

PABALATE-SODIUM FREE

Same formula as Pabalate, with sodium salts replaced by potassium salts (pink)

For the patient who requires steroids

PABALATE-HC

Pabalate with Hydrocortisone

In each light blue enteric-coated **PABALATE-HC** tablet:

Hydrocortisone	2.5 mg.
Potassium salicylate (5 gr.)	0.3 Gm.
Potassium para-aminobenzoate (5 gr.)	0.3 Gm.
Ascorbic acid	50.0 mg.

1. Ford, R. A., and Blanchard, K.: Journal-Lancet 78:185, 1958.

A. H. ROBINS CO., INC., Richmond 20, Virginia

PARAFLEX

Chlorzoxazone*

rapidly relieves both pain and stiffness . . . facilitates recovery

PARAFLEX provides effective skeletal muscle relaxation for 6 hours with a 1- to 2-tablet dose. It relieves painful muscle spasm and improves function in a wide variety of traumatic, arthritic, and rheumatic disorders. PARAFLEX is especially valuable when used in conjunction with physiotherapy and other rehabilitative procedures. Side effects are rare, almost never require discontinuance of therapy.

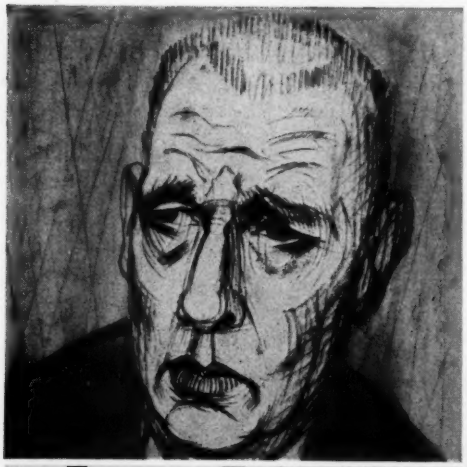
Dosage: ADULTS—1 to 2 tablets three or four times a day.
CHILDREN—½ to 2 tablets three or four times a day, depending on age and weight.
Supplied: Tablets, scored, orange, bottles of 50. Each tablet contains PARAFLEX, 250 mg.

*U. S. Patent No. 2,895,877

McNEIL LABORATORIES, INC • PHILADELPHIA 32, PA.

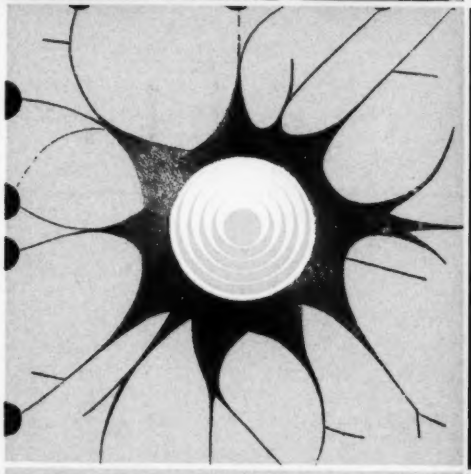
330A60

IN WHIPLASH INJURY



whenever depression complicates the picture

Tofrānil®
brand of imipramine HCl



hastens recovery

Geigy

In many seemingly mild physical disorders an element of depression plays an insidious etiologic or complicating role.

Because of its efficacy as an antidepressant, coupled with its simplicity of usage, Tofrānil is admirably adapted to use in the home or office in these milder "depression-complicated" cases.

It is always wise to recognize that depression may be an underlying factor... that Tofrānil may speed recovery in "hypochondriasis"; in convalescence when recovery is inexplicably prolonged; in chronic illness with dejection; in the menopausal patient whose emotional disturbances resist hormone therapy; and in many other comparable situations in which latent depression may play a part.

Detailed Literature Available on Request

Tofrānil, brand of imipramine hydrochloride, tablets of 25 mg. Ampuls for intramuscular administration, 25 mg. in 2 cc. of solution.

Geigy, Ardsley, New York

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In active people who won't take time to eat properly, MYADEC can help prevent deficiencies by providing comprehensive vitamin-mineral support. Just one capsule a day supplies therapeutic doses of 9 important vitamins plus significant quantities of 11 essential minerals and trace elements. MYADEC is also valuable in vitamin depletion and stress states, in convalescence, in chronic disorders, in patients on salt-restricted diets, or wherever therapeutic vitamin-mineral supplementation is indicated.

Each MYADEC Capsule contains: VITAMINS: Vitamin B_{12} crystalline—5 mcg.; Vitamin B_2 (riboflavin)—10 mg.; Vitamin B_6 (pyridoxine hydrochloride)—2 mg.; Vitamin B_1 mononitrate—10 mg.; Nicotinamide (niacinamide)—100 mg.; Vitamin C (ascorbic acid)—150 mg.; Vitamin A—(7.5 mg.) 25,000 units; Vitamin D—(25 mcg.) 1,000 units; Vitamin E (d-alpha-tocopheryl acetate concentrate)—5 I.U. MINERALS: (as inorganic salts) Iodine—0.15 mg.; Manganese—1 mg.; Cobalt—0.1 mg.; Potassium—5 mg.; Molybdenum—0.2 mg.; Iron—15 mg.; Copper—1 mg.; Zinc—1.5 mg.; Magnesium—6 mg.; Calcium—105 mg.; Phosphorus—80 mg. Bottles of 30, 100 and 250.

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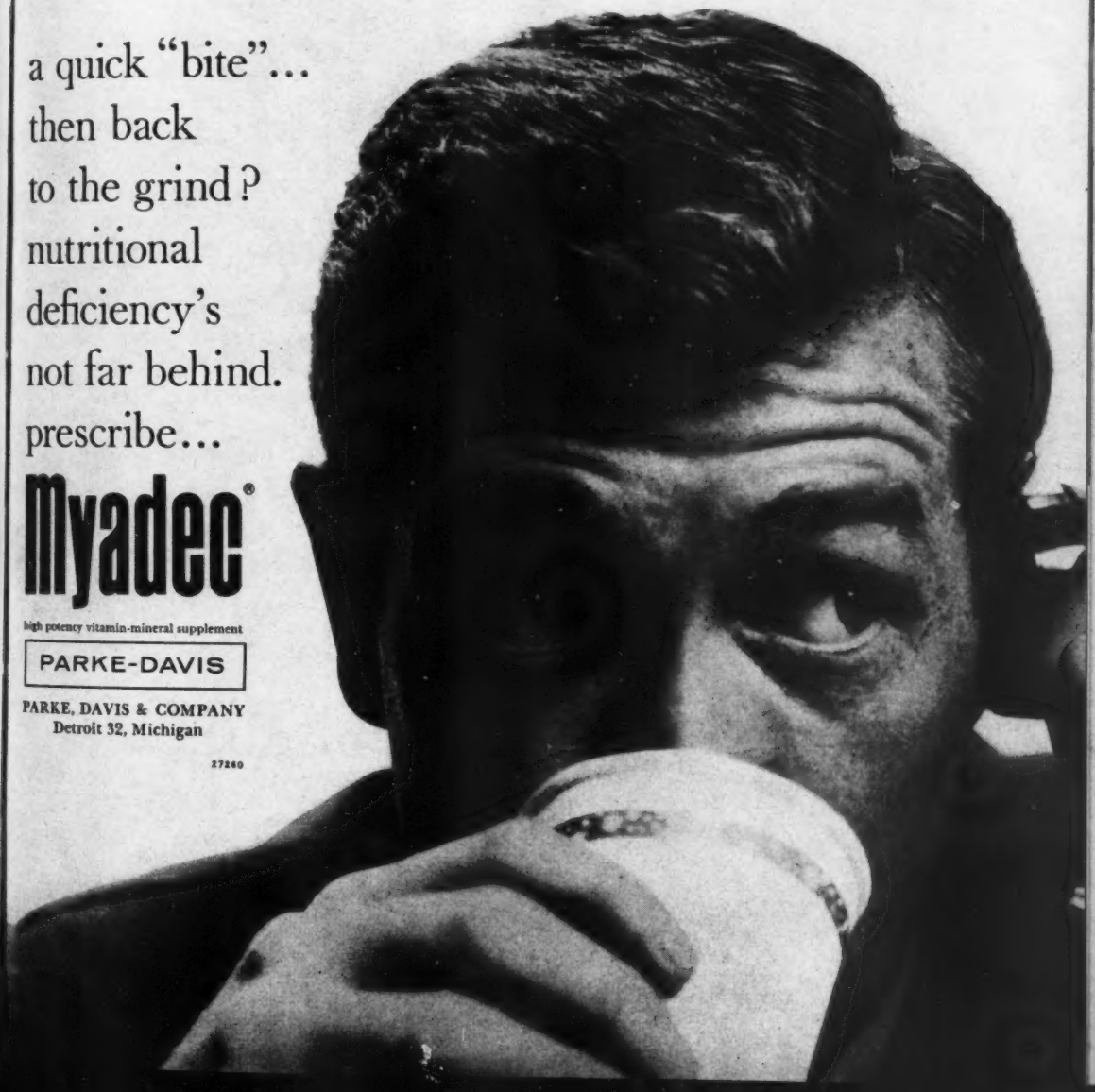

INCREASED LIFE EXPECTANCY FOR HYPERTENSIVES

"Life expectancy seems to be the one criterion that is most reliable and least questioned as a method of evaluating treatment for patients with elevated blood pressure."[1] "It is evident that effective therapy of hypertension will prolong the life of the patient by preventing the dreaded complications of this disease in the brain, the heart and the kidneys." "There is no doubt of the prolongation of life in group 3 and 4 (Keith-Wagener-Barker) by adequate antihypertensive treatment. Some authorities report a 50 per cent, five year survival ratio for treated patients with malignant hypertension as against a 1 per cent survival ratio for untreated patients."[2]

Evaluation based on life expectancy is extremely difficult because of the peril of maintaining an untreated control group.[1] The doctor, however, can evaluate the symptoms related to the elevated blood pressure. . . . We know that retinopathy may improve, the heart may be reduced in size, the electrocardiogram may improve and in favorable cases the blood urea nitrogen level may fall.[2] These are reasonably objective criteria on which to base one's evaluation of treatment.[1]

On the succeeding page is evidence that Unitensen included in any therapeutic regimen may improve the results in hypertension as measured by a regression of objective clinical changes in a substantial proportion of the patients treated.

1. Currens, J. H.: New England J. Med. *261*:1062, 1959.
2. Waldman, S., and Pelner, L.: Am. Pract. & Digest. Treat. *10*:1139, 1959.
3. Cohen, B. M.: paper presented at A.M.A. Convention, June, 1958.
4. Cohen, B. M.: paper presented at Indiana Acad. G. P., March, 1959.
5. Cohen, B. M.: Am. J. Cardiology *1*:748, 1958.
6. Kirkendall, W. J.: J. Iowa M. Soc. *47*:300, 1957.
7. Cherny, W. B., *et al.*: Obst. & Gynec. *9*:515, 1957.
8. Raber, P. A.: Illinois M. J. *108*:171, 1955.
9. McCall, M. L., *et al.*: Obst. & Gynec. *6*:297, 1955.
10. Finnerty, F. A.: Am. J. Med. *17*:629, 1954.

Unlike diuretics or ganglionic blocking agents, Unitensen lowers blood pressure through widespread vasorelaxation. Normal vasomotor responses are not altered, and there is no venous pooling with resulting postural hypotension.[3-5] Through alleviation of cerebral vasospasm, Unitensen promotes cerebral blood flow and oxygen utilization.[6-9] Furthermore, Unitensen increases cardiac efficiency, improves renal function and tends to arrest the progress of vascular damage.[3, 4, 10]

Progress of Objective and Subjective Symptoms in Grades III and IV Hypertension Following Treatment with Unitensen and Unitensen-R

Observations in Patients* Treated up to 2 Years

Observations in Patients* Treated up to 3½ Years

The Course of Subjective Symptoms

Symptom	Number**	Improved	% Improved	Number**	Improved	% Improved
Headache	27	21	77.7	43	38	88.0
Palpitation	20	13	65.0	29	19	65.5
Angina	15	9	60.0	21	16	76.0
Dyspnea	17	8	47.0	27	14	51.0

Objective Changes Following Treatment

Finding	Number**	Improved	% Improved	Number**	Improved	% Improved
Funduscopic Changes	41	24	58.5	59	38	66.0
Enlarged Heart	20	13	65.0	35	23	65.7
Abnormal ECG	37	10	27.0	45	25	55.5
Proteinuria	31	12	38.7	43	27	62.7
Nitrogen Retention	17	6	35.2	28	10	35.7

Left hand charts from Clinical Exhibit "The Ambulatory Patient with Hypertension" presented AMA Convention, San Francisco, June 22-27, 1958, by B. M. Cohen, M.D.

Right hand charts include patients previously reported who had been continuously maintained on Unitensen and Unitensen-R, plus additional patients later added to the study. From Clinical Exhibit "The Office Diagnosis and Treatment of the Patient with Hypertension" presented American Academy of General Practice, Indianapolis, March 18-19, 1959, by B. M. Cohen, M.D.

*All patients in this study were initially classified as Smithwick Grades III and IV.

**Expressed as the number of patients exhibiting the symptom recorded.

UNITENSEN®
Each tablet contains: Cryptenamine (tannates) 2.0 mg.

UNITENSEN-PHEN®
Each tablet contains: Cryptenamine (tannates) 1.0 mg., Phenobarbital 15 mg.

UNITENSEN-R®
Each tablet contains: Cryptenamine (tannates) 1.0 mg., Reserpine 0.1 mg.

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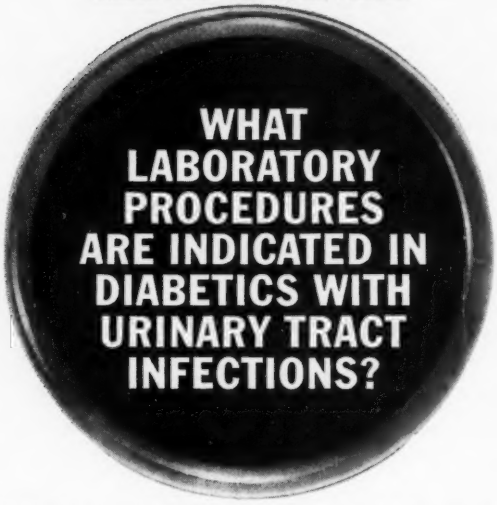

A urine culture is absolutely essential in the diabetic suspected of having a urinary tract infection since such infection is not always accompanied by pyuria. It is also essential to keep the urine free from sugar—as shown by frequent urine-sugar tests—for successful therapy.

Source: Harrison, T. R., *et al.*: Principles of Internal Medicine, ed. 3, New York, McGraw-Hill Book Co., 1958, p. 620.

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color-calibrated

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the *standardized* urine-sugar test for reliable quantitative estimations

Urinary tract infections are about four times more frequent in the diabetic than in the non-diabetic. The prevention and treatment of urinary tract infections, as well as the avoidance of other complications of diabetes, are significantly more effective in the well-controlled diabetic. The patient should be impressed repeatedly with the importance of continued daily urine-sugar testing—especially during intercurrent illness—and warned of the consequences of relaxed vigilance.

"urine-sugar profile" With the new *Graphic Analysis Record* included in the CLINITEST Urine-Sugar Analysis Set (and in the tablet refills), daily urine-sugar readings may be recorded to form a graphic portrayal of glucose excretion most useful in clinical control.

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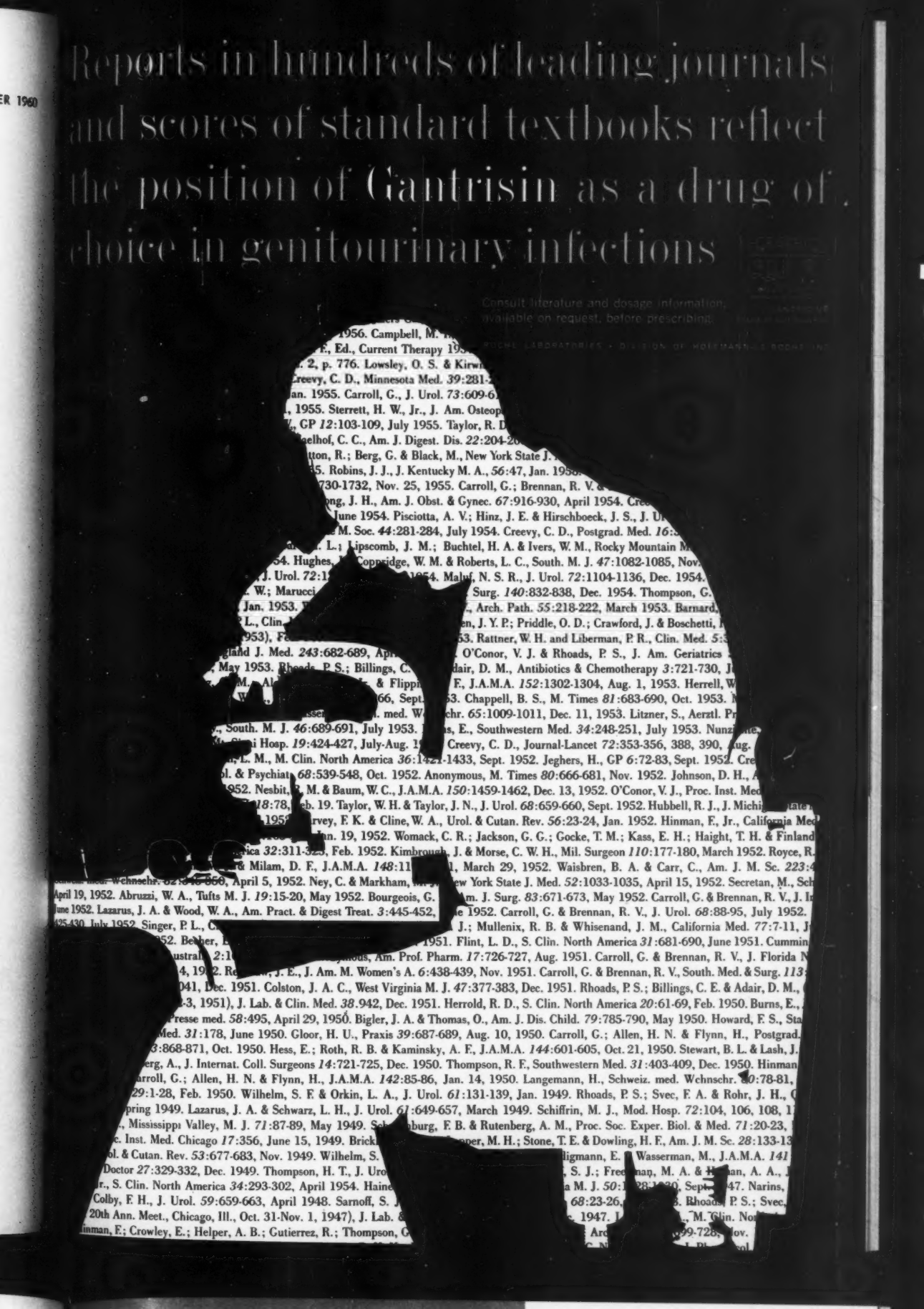

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1,928 published cases in the two years since TAO was released for general use show:

94.3% effectiveness in respiratory infections (617 cases including tonsillitis, staphylococcal and streptococcal pharyngitis, bronchitis, infectious asthma, broncho-pneumonia, lobar pneumonia, bronchiectasis, lung abscess, otitis.) **You can count on TAO.**

92% effectiveness in skin and soft tissue infections (900 cases including pyoderma, impetigo, acne, infected skin disorders, wounds, incisions and burns, furunculosis, abscess, cellulitis, chronic ulcer, adenitis.) **You can count on TAO.**

87.1% effectiveness in genitourinary infections (349 cases including urethritis, cystitis, pyelitis, pyelonephritis, orchitis, pelvic inflammation, acute gonococcal urethritis, lymphogranuloma venereum.) **You can count on TAO.**

75.8% effectiveness in diverse infections (62 cases including fever of undetermined origin, peritoneal abscess, osteitis, periarthritis, septic arthritis, staphylococcal enterocolitis, gastroenteritis, carriers of staphylococci.) **You can count on TAO.**

95.6% of 1,928 cases free of side effects —in the remaining 4.4%, reactions were chiefly mild gastrointestinal disturbances which seldom necessitated discontinuance of therapy.

* In 884 of 1,928 cases the causative organisms were mostly staphylococci. The majority of clinical isolates were found to be resistant to at least one of the commonly used antibiotics and many patients had failed to respond to previous therapy with one or more antibiotics. **TAO proved 93.4% effective in these 884 cases.**

Complete bibliography available on request.

DOSAGE: varies according to severity of infection. Usual adult dose—250 to 500 mg. q.i.d. Usual pediatric dose: 3-5 mg./lb. body weight every 6 hours.

NOTE: In some children, when TAO was administered at considerably higher than therapeutic levels for extended periods, transient-jaundice and other indications of liver dysfunction have been noted. A rapid and complete return to normal occurred when TAO was withdrawn.

SUPPLY: TAO CAPSULES—250 mg. and 125 mg., bottles of 60. TAO ORAL SUSPENSION—125 mg. per 5 cc. when reconstituted, palatable cherry flavor, 60 cc. bottles. TAO PEDIATRIC DROPS—100 mg. per cc. when reconstituted, flavorful; special calibrated dropper, 10 cc. bottles. INTRAMUSCULAR or INTRAVENOUS—10 cc. vials, as oleandomycin phosphate.

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important new therapy in Peptic Ulcer

cessation of all symptoms and complete healing in 70 out of 78 cases as reported in Postgraduate Medicine (Oct.) 1959

"...chymotrypsin offers a new approach to the treatment of peptic ulcer."

In 54 cases, most of them hospitalized, in which chymotrypsin (Chymar) was used in conjunction with other agents "All of the symptoms disappeared and *complete healing of* the ulcer occurred in 49 (90.7 per cent) of the 54 cases . . . " Average time for cessation of symptoms . . . 6 days; for complete healing . . 36 days; average follow-up period . . . 12 months. *In 24 cases* in which Chymar was used alone, "Cessation of all symptoms and *complete healing occurred in 21 (87.5 per cent)* of the 24 cases . . ." Average time for cessation of symptoms . . . 5.8 days; for complete healing . . . 24 days; average follow-up period . . . 25.5 months.

Conclusions: "Because of the excellent results obtained in 78 cases of peptic ulcer . . . I strongly recommend its use as a most valuable adjunct in the treatment of this disease."*

*Mozan, A. A.: Postgraduate Med. 26:542, 1959

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chymotrypsin *Buccal/Aqueous/Oil*

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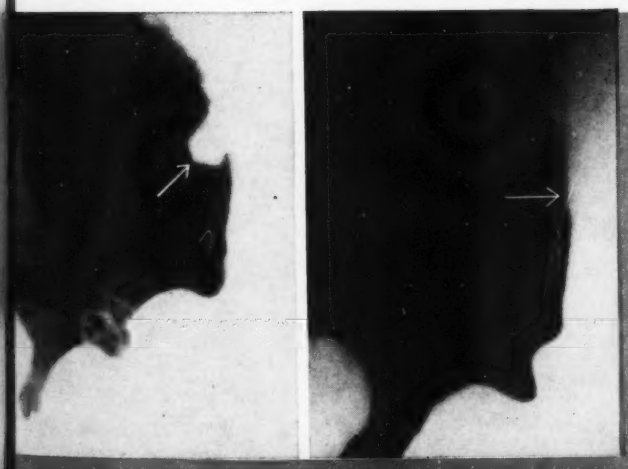

Fluoroscopic roentgenogram made on January 26, 1957 shows a large niche on the upper third of the lesser curvature.

Roentgenogram made on February 23, 1957 shows only a slight indentation on the lesser curvature.

CHYMAR Buccal—Crystallized chymotrypsin in a tablet formulated for buccal absorption. Bottles of 24 tablets. Enzymatic activity, 10,000 Armour Units per tablet.

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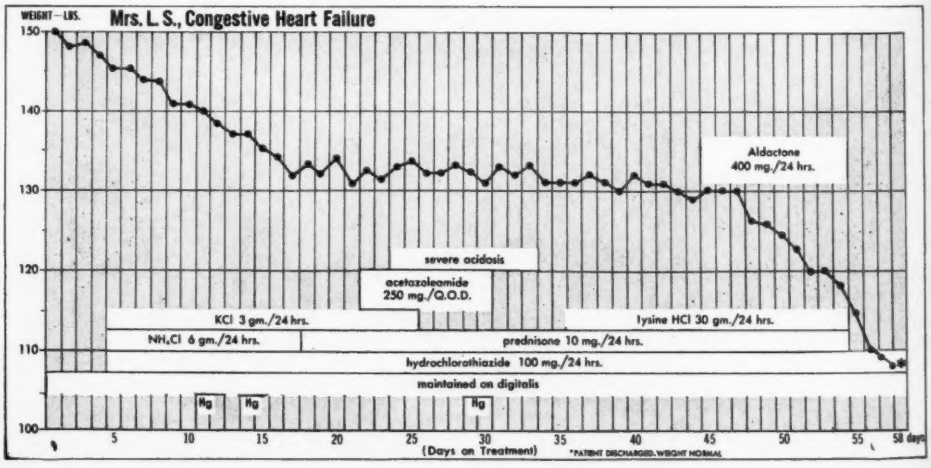

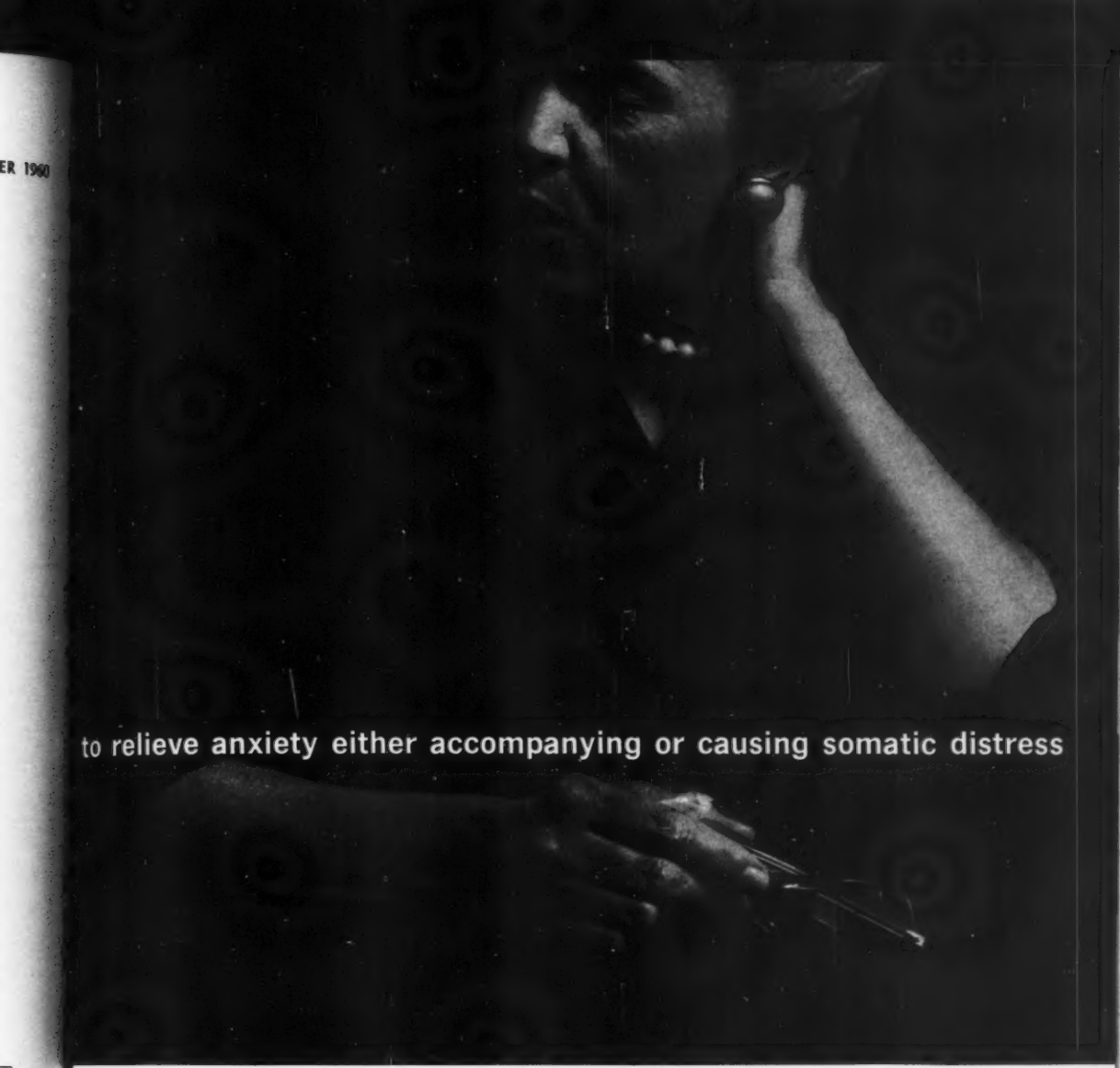

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brand of trifluoperazine

- **Prompt control of the underlying anxiety.** Beneficial effects are often seen within 24-48 hours.
- **Amelioration of somatic symptoms.** Marx[1] reported from his study of 43 office patients that 'Stelazine' "appeared to be effective for patients whose anxiety was associated with organic—as well as functional disorders."
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Optimal dosage: 2-4 mg. daily. Available as 1 mg. and 2 mg. tablets, in bottles of 50 and 500.

N.B.: For further information on dosage, side effects, cautions and contraindications, see available comprehensive literature, *Physicians' Desk Reference,* or your S.K.F. representative. Full information is also on file with your pharmacist.

1. Marx, F.J., in *Trifluoperazine: Further Clinical and Laboratory Studies*, Philadelphia, Lea & Febiger, 1959 p. 89
2. Winkelman, N.W., Jr.: *ibid.*, p. 78.

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adult stable diabetics

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sulfonylurea failures

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"Most mild diabetic patients were well controlled on a biguanide compound [DBI], and such control was occasionally superior to that of insulin. This was true regardless of age, duration of diabetes, or response to tolbutamide."[2]

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Among those diabetics who responded to tolbutamide initially and became secondary failures **DBI "gave a satisfactory response in 55%."**[4]

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1. Walker, R. S.: Brit. M. J. 2:405, 1959.
2. Odell, W. D., et al.: A.M.A. Arch. Int. Med. 102:520, 1958.
3. Pearlman, W.: Phenformin Symposium, Houston, Feb. 1959.
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6. Miller, E. C.: Phenformin Symposium, Houston, Feb. 1959.
7. Krall, L. P.: Applied Therapeutics 2:137, 1960.

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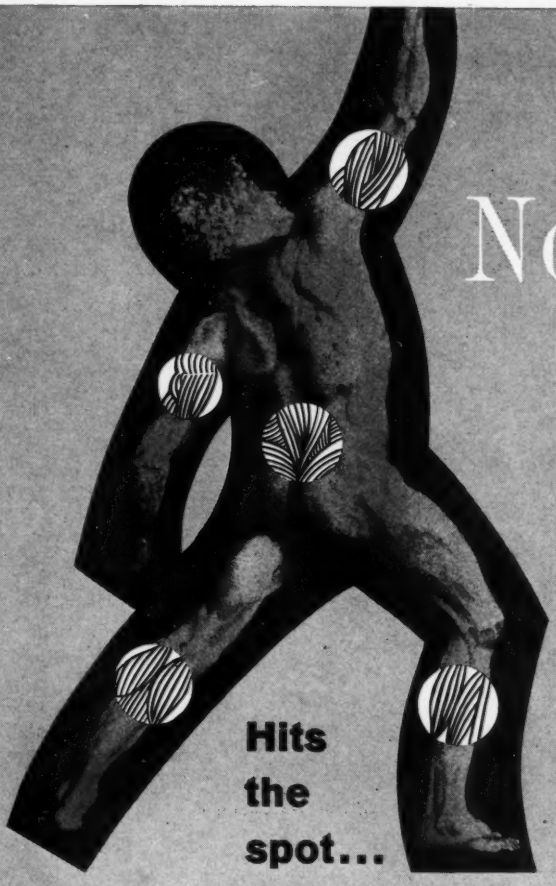

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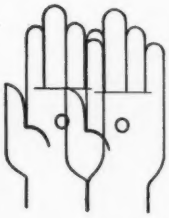

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1. *Jones, G. S. and Smith, F.: Am. J. Obstet. Gynecol., Vol. 67, No. 3, 628-633, 1954.*
2. *Jones, Scott S.: Northwest Medicine, Vol. 54, 1253-1254, 1955.*
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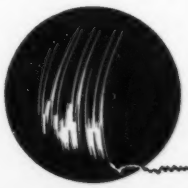

in vivo measurement of LUTREXIN on contracting uterine muscle

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Southern Medical Journal

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September 1960

Correction of Hypospadias*

HAROLD P. McDONALD, M.D., WILBORN E. UPCHURCH, M.D., and CARLOS L. CELAYA, M.D., *Atlanta, Ga.*

The authors emphasize the important steps in the correction of this congenital anomaly. They have used steps from two classical procedures advanced in the past for this reconstructive surgery.

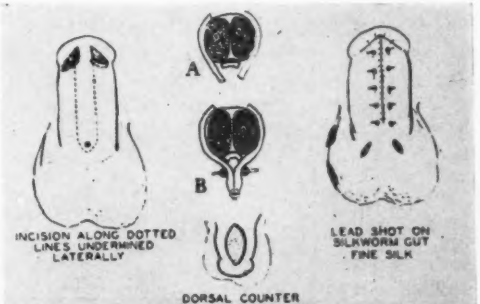
HYPOSPADIAS is a common congenital anomaly, characterized by ventral displacement of the urethral meatus and by a chordee which causes a downward curvature of the penis, particularly noticeable when erection occurs. This chordee is produced by a fibrous band or bands on the ventral surface of the penis which displaces the corpus spongiosum, either entirely or almost entirely. The fibrous tissue acts to produce a downward curvature of the penis which is markedly exaggerated upon erection. The urethral meatus is frequently quite narrow and the prepuce is usually hooded and almost entirely confined to the dorsal portion of the penis. This excess portion of prepuce is of great use in repair of hypospadias and particularly in reconstruction of the urethra.

Correction of hypospadias is divided into two stages,—the first is concerned with the straightening of the penis and elimination of the chordee, the second is the formation of an adequate urethra so the individual is able to void properly. Early correction of the chordee allows proper development of the corpora cavernosa, so the organ is straight upon erection and so sexual function may therefore be satisfactory. Furthermore, with the normal urethra terminating at, or near the end of the penis insemination is possible.

The first description of an operation for correction of hypospadias was made by Duplay of France who, in 1880, described a surgical treatment for hypospadias and epispadias. Since that time and until about 1940, the Duplay operation or a modification of it was the operation of choice in attempted correction of hypospadias. Denis Browne[1] of England, in 1936, published his procedure, "An Operation for Hypospadias," in which he described an operation for correction of hypospadias that was a major departure from previously described procedures.

Since the first description of the Denis Browne procedure for construction of an urethra, others have used it or slight modifications of it with notable success. Among these are Burns,[2] Campbell,[3] Johanson,[4] Davis[5] and others. The technic consists essentially of the formation of the new urethra by a buried skin strip which is covered over by a flap on either side secured by sutures without tension (Fig. 1). Nesbit, Butler and Whitaker,[6] in 1950,

FIG. 1


A
B
INCISION ALONG DOTTED LINES UNDERMINED LATERALLY
LEAD SHOT ON SILKWORM GUT FINE SILK
DORSAL COUNTER INCISION
Mr. Denis Browne's urethroplasty for hypospadias

Illustrations taken from Mr. Denis Browne's original article showing steps in the Denis Browne urethrosplasty. Cross sections A and B demonstrate closure of skin flaps over buried strip of skin.

*Read before the Section on Urology, Southern Medical Association, Fifty-Third Annual Meeting, Atlanta, Ga., November 16-19, 1959.

demonstrated in experimental animals that strips of skin buried in the abdominal wall of the rat developed complete circumferential epithelization and that a tube was formed.

We have used the Duplay method for the correction of hypospadias with slight modifications with quite satisfactory results. Since the publication of the method of Denis Browne, we have used this method in the patients where insufficient amount of dorsal foreskin was present to permit formation of a tube on the ventral surface of the penis according to the method of Duplay.

Classification and Treatment of Hypospadias

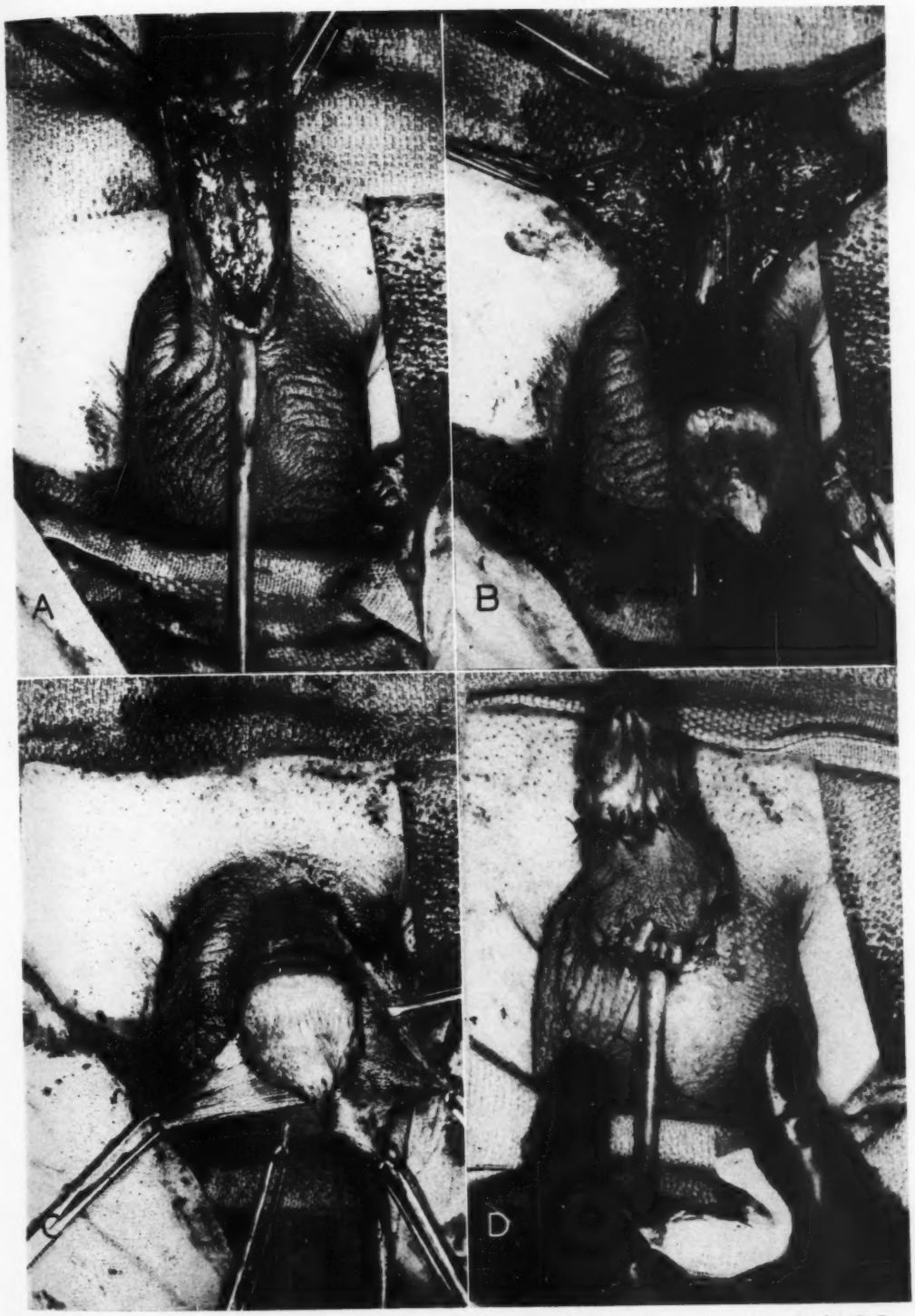
Hypospadias may be divided into two main classifications, mild, and moderate to severe.

A *mild* degree of hypospadias is one in which the amount of chordee is slight and is insufficient to interfere with a satisfactory erection, and the penis is relatively straight. The ventral displacement of the urethral meatus is slight. This type of hypospadias is often referred to as glandular or first degree hypospadias and very little treatment or surgery is needed for correction. The narrow urethral meatus, which is almost always present, can usually be improved by an incision in the dorsal aspect or roof of the urethra. This in effect is a dorsal meatotomy and provides a wide meatus at the end of the penis. In some instances a small amount of excess mucous membrane is excised from the roof of the new urethral meatus after making the dorsal meatotomy. It is particularly important to be sure that chordee is absolutely minimal before circumcision or removal of excess dorsal prepuce is done in correction of such mild hypospadias.

The *moderate* to *severe* degrees of hypospadias require two operations for correction. The first operation is for elimination of the chordee and is preferably done at an early age of life. It is recommended that this first stage procedure be done by the time the child reaches 18 months to two years of age. It is essential that at this operation all fibrous bands, restricting the straightening of the penis on erection, be divided and removed. A transverse incision is made on the ventral surface of the penis just distal to the urethral meatus and is carried down to the corpora cavernosa incising the fibrous bands found on the capsule of the corpora cavernosa and immediately between the corpora cavernosa. These bands usually fan out proximally and all should be sought out, cut and excised, so that traction on the glans penis allows complete extension of the organ in a straight and satisfactory manner. The incision is carried laterally and downward on either side of the shaft of the penis and the urethra is freed from all the adherent bands so that upon completion of the straightening portion of this first stage operation, it will be noted that the urethral opening is much further back from the glans than at the beginning of the operation. As a matter of fact, the urethral meatus will have changed from a position just immediately back of the glans to a position more properly described as penoscrotal in location. The entire ventral surface of the penis will at this point be completely denuded of skin and subcutaneous tissue. A transverse incision is then made immediately back of the corona on the dorsal aspect of the penis and the entire subcutaneous tissue comprising the excess redundant prepuce is freed up and held upward more or less as a tent above the penis. At this point this skin flap may be split in the middle or a buttonhole incision, as recommended by Nesbit,[6] may be made through the midportion of this flap, so that with the glans penis drawn through this buttonhole the excess of the prepuce is carried to the ventral surface of the penis where it is sutured to the exposed surface of the corpora cavernosa, thus covering all the raw area left by the excision of the chordee producing fibrous tissue (Fig. 2). At this stage the operator may be tempted to trim away a small bit of the excess preputial skin on the lateral margins of the penis, but to do this would be a mistake as it is wise to preserve all of the apparently excess skin so it may be used later in constructing an urethra. Customarily we place a suture through the tip of the glans penis and exert a slight upward pull on the penis by stitching this suture to the abdominal skin surface above the symphysis pubis. This suture is removed with the first change of dressings. The urethral meatus is dilated or meatotomy is performed and a retention catheter is inserted through the urethra and a mild pressure dressing is applied, after sprinkling a small amount of sulfanilamide powder in and around the wound.

For two days previous to the operation and for one week following the operation, it has

FIG. 2



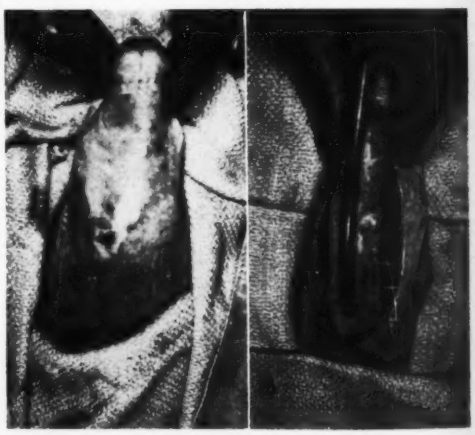
Steps illustrating first stage operation for hypospadias. (A) Transverse ventral incision and removal of fibrous bands. (B) Development of dorsal skin flap from prepuce. (C) Transfer of dorsal flap to ventral surface after buttonhole incision in flap. (D) Dorsal flap sutured to ventral surface of penis.

been our custom to administer Stilbestrol in 1 to 5 mg. doses by hypodermic twice daily. This administration of Stilbestrol effectively inhibits erections during the immediate postoperative period and contributes, in our opinion, to the almost complete *per primum* healing that we have encountered in our cases. The dressing is removed after about five days and the patient is given hot tub baths daily. The hospital stay for this first stage operation averages six to eight days. The time interval between the first and second stage operations should be six months or more. If the first operation is performed at the age of 18 months or two years, the second operation should be performed by the age of five so the child may be able to void normally about the time he begins to enter school. If the boy is older when the first operation is done, it is wise to wait at least six months before the second stage operation to permit maximum softening and pliability of the skin and subcutaneous tissues of the penis. Before the second operation for the formation of the urethra, we customarily administer Stilbestrol as previously to lessen or prevent erections postoperatively.

Diversion of the urine at the time of the second stage is an essential part of the success in this operation. While some surgeons advocate diversion of the urine by a perineal urethrostomy, it has been our custom to divert the urine by suprapubic cystopuncture and have found this uniformly successful and quite easy to do. The cystopuncture is done after first distending the bladder to at least 300 cc. The skin is incised just above the symphysis pubis, the incision being carried down through the rectus fascia. A spear point No. 11 blade knife is used. A No. 18 or 20 ordinary trocar is used to enter the distended bladder and a No. 18 or 20 balloon catheter inserted. When counter drainage is no longer needed, simple removal of the catheter is followed by closure of the suprapubic puncture wound within 24 hours in practically every instance.

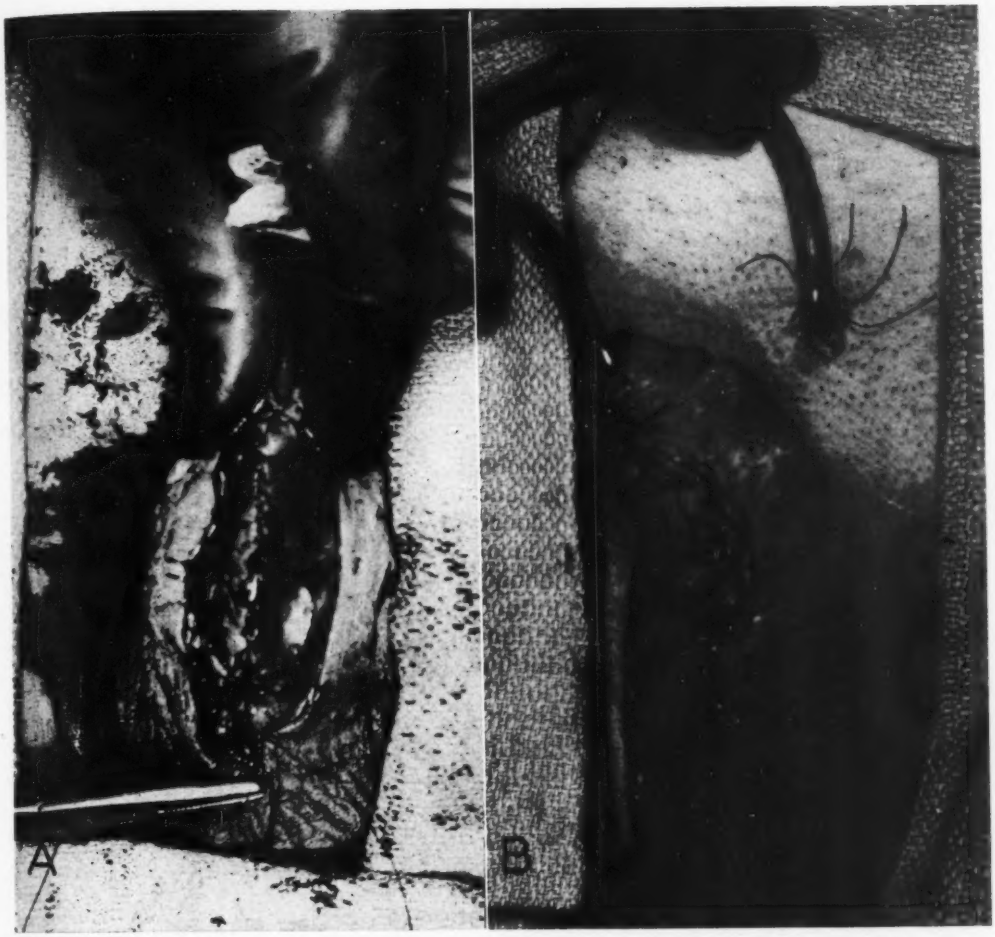
At the second stage or procedure for the formation of the urethra, if a sufficient amount of the excess dorsal portion of the preputial skin has been transferred to the ventral surface to allow a Duplay or tube-forming operation, this is preferred over the Denis Browne procedure. The incision is made around the urethral opening and carried up the ventral side of the penis to the glans penis on either side (Fig. 3). The edges of the incision are undermined on both sides so as to leave approximately one half of the strip attached to the ventral surface of the penis. The lateral edges are undermined also, these lateral edges to be brought across the midline without tension. The urethra is formed over a small catheter, usually a No. 14 or 16 balloon type catheter. This allows smooth approximation of the edges and formation of the uniform caliber urethra. We have used 0000 catgut in a continuous running stitch in almost every case during the past ten years, and it is our belief that this type running suture permits a better approximation with the elimination of pressure suture points than when interrupted sutures are employed. A second row of continuous fine atraumatic catgut is placed in the subcutaneous tissue approximating this tissue over the newly formed urethral tube. A third layer is then formed by mattress sutures in the skin, being careful to prevent inversion of skin edges (Fig. 4). A dorsal incision through the skin and subcutaneous tissue of the penis is made occasionally but only in such instances where there appears to be excessive tension on the third layer of skin sutures or the mattress sutures. This is a modification borrowed from the Denis Browne operation. Careful hemostasis is practiced throughout, and a bandage making slight pressure is applied upon completion, again after first sprinkling the wound with sulfanilamide powder. Antibacterial

FIG. 3



Second stage hypospadias operation. (A) Showing absence of chordee. (B) Line of incision for formation of urethra.

FIG. 4



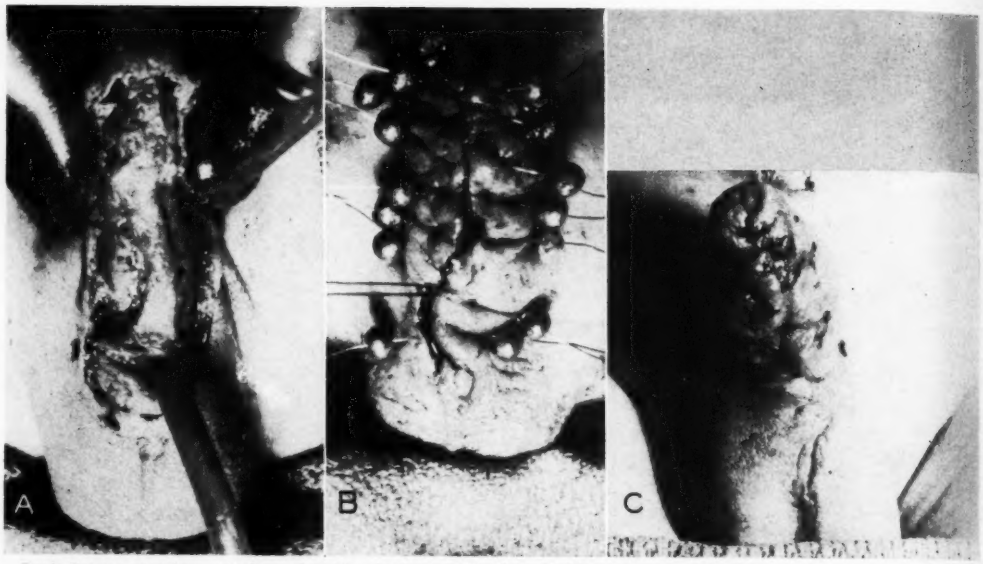
Further steps in second stage of correction for hypospadias. (A) Tube formed by means of skin flap; continuous fine catgut sutures. (B) Final skin closure with mattress sutures. Also shown is the suprapubic counter drain.

medication is administered immediately before and after operation. At the present time we prefer dihydrostreptomycin and penicillin in the form of "Combiotic" administered daily. The daily administration of Stilbestrol is continued and usually within five or six days the dressing is off, and the patient is put in a hot tub of water daily or twice daily. The indwelling catheter is left in, usually through the healing period; however, it may be removed at any time it becomes evident that inflammation is present, or there is swelling, or pressure that might interfere with healing.

The suprapubic counter drainage tube is maintained for a period of several weeks, or until the urethra has healed sufficiently to permit the voiding of urine. In our experience this has averaged about four weeks. The urethral retention catheter may be removed after the first few days or a week, and may be replaced during the third week for a few days. In our experience this indwelling urethral catheter has been of help in the formation of the urethra and assists in healing by molding the urethra and elimination of suture pressure points. We believe the administration of Stilbestrol and antibacterial medication, as well as the hot tub baths, are important to the success of this procedure.

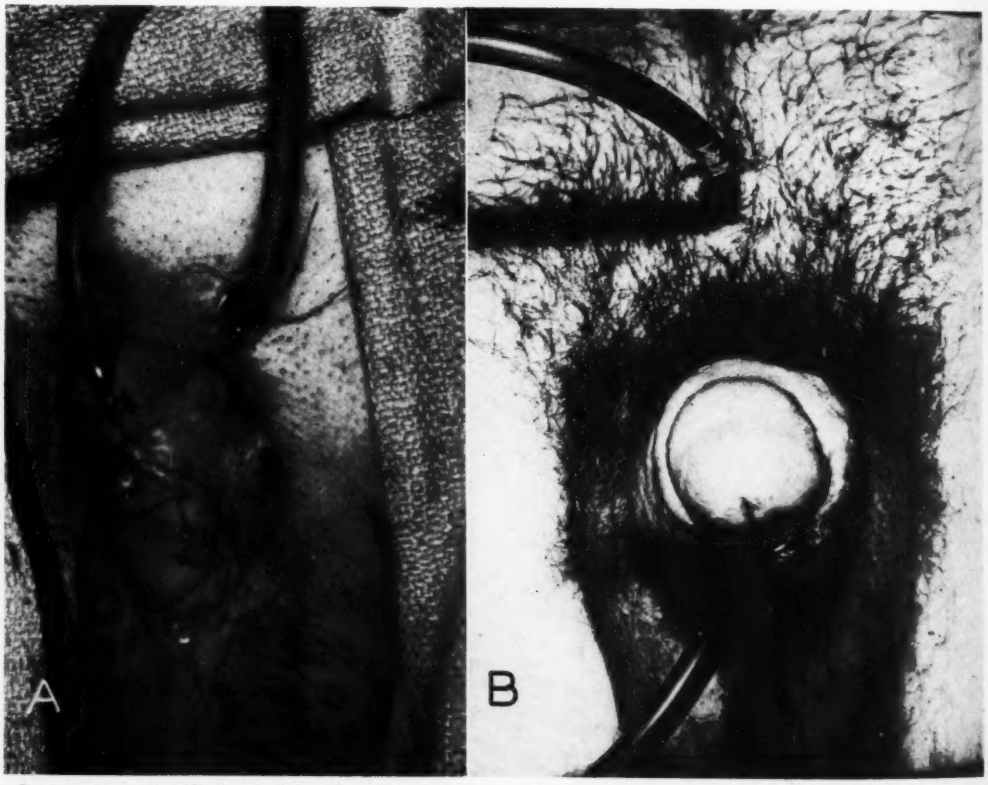
At times, after completion of the first stage

FIG. 5



Denis Browne procedure. (A) Line of incision for buried skin flap. (B) Mattress sutures with fine steel wire. (C) Result: three weeks postoperative.

FIG. 6



Postoperative result. (A) At completion of operation. (B) Four weeks later prior to removal of suprapubic and urethral catheters.

operation or procedure for the correction of chordee, it is found that there is insufficient skin for this formation of the urethra by the Duplay method. In this instance, it has been our practice to use the Denis Browne procedure with the slight modification that an indwelling urethral catheter be used and the suprapubic cystopuncture be done as described before (Fig. 5). It is our belief that the indwelling urethral catheter so used aids in the formation of the circular tube, and in some measure reduces the amount of the swelling, bleeding and exudation of serum. It may be said that the use of the indwelling urethral catheter is unsound, but we have not found it so and, as a matter of fact, have been impressed with its aid in the second stage operation in the formation of the new urethra. The use of a tube in this manner may be said to be similar to that of the intubated ureterotomy procedure described by David M. Davis (Fig. 6).

It is very important to eliminate tension on sutures and to avoid undue tightness in tying sutures. The use of fine chromic catgut on atraumatic needles, meticulous attention to the control of hemorrhage and the use of bandage dressings making mild pressure are all important points in technic.

The suprapubic cystopuncture is preferred to the perineal urethrostomy tube for urinary counter drainage. Since adopting this technic, we have had no failures in the last 18 operations for hypospadias.

Summary

Correction of hypospadias should be begun early, preferably by the age of two years. The first stage or correction of the chordee consists of complete elimination of fibrous bands restricting the ventral elongation of the penis. A minimum of six months should elapse before the second stage procedure for the formation of the urethra is attempted. The use of Stilbestrol pre- and postoperatively to eliminate troublesome erections is recommended. Formation of the urethra over a small urethral catheter is recommended and hot tub baths early during the postoperative period encourage rapid healing.

Avoidance of suture pressure points and elimination of multiple knots in the subcutaneous tissues by the use of a continuous suture of fine chromic catgut on atraumatic needles are recommended. Care to avoid undue pressure or tension is an essential factor in encouraging primary healing. A modified Duplay technic as well as the Denis Browne technic for repair of hypospadias are both recommended and it is essential that a new urethra be of adequate caliber.

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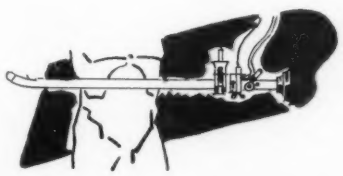

The Comparative Accuracy in Localization of Brain Tumors by Air Contrast Radiography, Angiography, and Electroencephalography

ROBERT L. GREEN, M.D., and EWALD W. BUSSE, M.D.,† *Durham, N. C.*

The localization of intracranial tumors still presents a problem in diagnosis. The electroencephalogram has been shown to be the most valuable screening method in the hands of these authors. Furthermore, its ease of repetition for diagnosis in suspected disease offers still a greater place in diagnosis.

DURING THE PAST HALF CENTURY tremendous advances have been made in the early diagnosis and surgical management of tumors of the brain. The improvements in neurosurgical management have, to a large extent, been dependent on or related to the development of better technics for more accurate localization of the tumors. In 1905, Cushing[1] stated that, "The diagnosis of intracranial growth is usually simple, but the recognition of its exact location is much more puzzling." Localization of tumor sites, based on detailed neurologic examination alone, often not only proved embarrassing to the neurosurgeon but fatal to the patient.

The first revolutionary advancement in the technic of the localization of tumors by radiography came in 1918 and 1919, when Dandy[2,3] described pneumoventriculography and pneumoencephalography. It took the next 10 or 15 years for these diagnostic procedures to become accepted and universally used.

The second advance occurred in 1927, when Moniz[4] introduced cerebral angiography, the injection of contrast media into the carotid arteries to outline the intracranial blood vessels as shown by a radiograph.

The next valuable advance took place in 1929, when Berger[5] published the first important work on electroencephalography. This was greeted with great skepticism; however, during subsequent years the electroencephalogram, like air studies and angiography, has become more widely accepted and used.

Each procedure has its own special technical difficulties and shortcomings.[6] These include herniation in the presence of increased intracranial pressure, edema, hemorrhage and malacia resulting from ventriculography or pneumoencephalography. There may be reactions to the contrast media during angiography, and during electroencephalographic examinations poor cooperation with artifact due to muscle and movement in the confused or obtunded patient is also encountered.

Ventriculography, pneumoencephalography, angiography and electroencephalography are by no means all of the diagnostic tests we have available, but when combined with a good clinical history and detailed neurologic examination these procedures appear more fruitful and, at present, are the most widely accepted and used.

The methods for doing contrast radiography and angiography are fairly well standardized. However, there are many technics for EEG. recordings, some apparently more adequate for localization than others. If we are going to assess without bias the value of various EEG. technics, comparative studies must be made.

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Material and Methods

This study consists of 99 proved cases of brain tumor that have been hospitalized and treated at the Veterans Administration Hospital, Durham, since it opened in 1953. All patients had brain tumors proved either at operation or upon postmortem examination. Tumors of all types and intracranial localizations make up the group. They include 57 gliomas, 11 meningiomas, 17 vascular tumors, 13 metastatic tumors, and 1 pinealoma. (Forty-one of the patients died at our hospital, and detailed postmortem examinations were done.) The ages of the 99 patients varied from 24 to 70 years, and a total of 304 preoperative diagnostic procedures were done. These included 48 ventriculograms, 47 pneumoencephalograms, 110 angiograms and 99 electroencephalograms.

The ventriculograms, pneumoencephalograms and angiograms were performed in the standard manner. The EEG. studies were done on an 8 channel Grass III-D machine. Seventeen electrodes were used for the routine EEG.'s, and 21 electrodes were used for special studies. In addition, nasopharyngeal electrodes were used for more adequate localizing studies on 5 patients. The electrodes were placed according to the International Standard 10-20 System,[7] and the recordings were essentially bipolar.

During the evaluation of these patients, many of the diagnostic procedures were repeated two or more times on the same patient. In such instances, if the initial diagnostic procedure was reported as abnormal, and the subsequent study was done to better localize the abnormality, together they are counted as one test. However, if the initial procedure was reported as normal or unsatisfactory and a second procedure yielded the abnormality, these are counted as two separate tests. (In none of the diagnostic tests was an initial abnormal report given and followed later by a normal report on repeated examination.) When both initial and subsequent examinations are reported as negative or noncontributory, these also are counted as one test.

The reported localizations for each diagnostic procedure were evaluated as follows: (1) *Exact localization*—a craniotomy at the indicated site would reveal not less than one half of the tumor. (2) *Partial localization*—craniotomy at the indicated site would reveal at least one fifth but less than one half of the tumor. (For statistical purposes the classifications of exact and partial were combined.) (3) *Lateralizing*—diagnostic procedure reported as abnormal but only localized to hemispheral involvement. (4) *Abnormal but nonfocal*—procedure reported as indicating an abnormality but no localizing signs. (5) *False localization.* (6) *Negative or noncontributory* procedure.

Results. An analysis of the four procedures performed on the 99 tumor patients revealed the following accuracy as summarized in table 1.

TABLE 1

99 PATIENTS

Procedure	*Total Number*	*Number Accurate*	*Per Cent Accuracy*
Ventriculography	48	39	81.3%
Pneumoencephalography	43	26	60.5%
Angiography	82	53	64.6%
EEG.	80	58	72.5%

Comments on Table 1: The difference in the numbers listed for each procedure is accounted for by the fact that many patients in the total group had only one or two of the specified tests. In such instances this appeared to be all that was necessary for a diagnosis with accurate localization.

In addition to this evaluation of the entire tumor group, there were 40 patients upon whom all three major diagnostic procedures were performed one or more times (i.e., air studies, angiography and EEG.'s). Without corrections for statistical purposes, it will be noted that 173 diagnostic procedures were performed on these 40 patients. These include 60 EEG.'s, 59 angiograms and 54 air studies. From this, one might conclude that these 40 patients represent some of the more difficult diagnostic problems. The results in this group are summarized in table 2.

TABLE 2

40 PATIENTS

Procedure	*Total Number*	*Number Accurate*	*Per Cent Accuracy*
Ventriculography	19	13	68.4%
Pneumoencephalography	33	18	54.6%
(Ventriculo- and pneumo-combined)	52	31	59.6%
Angiography	44	26	59.1%
EEG.	46	33	71.7%

Comments on Table 2: For the air studies either a ventriculogram or a pneumoencephalogram was done, and on 6 patients both studies were performed. After correcting for repeated examinations, a total of 19 ventriculograms and 33 pneumoencephalograms were done. The ventriculograms were 68.4% accurate, and the pneumoencephalograms were 54.5% accurate with a combined accuracy of 59.6 per cent.

Forty-four angiograms yielded a 59.1% accuracy. Forty-six electroencephalograms were done with a 71.7% accuracy.

Discussion

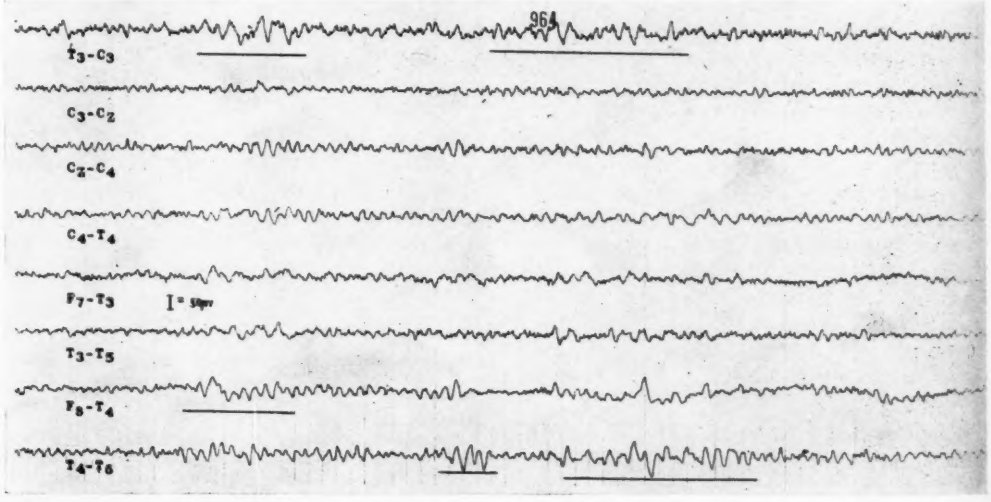
The data presented demonstrate the relative merits of electroencephalography as a localizing procedure in patients with brain tumors. Although the percentage of accuracy for the EEG. in those patients with all three procedures is slightly higher than for radiographic technics, this may be of limited clinical importance.

Neurosurgeons are not willing to perform a craniotomy on the basis of electroencephalographic abnormalities alone. Even when the clinical history and/or neurologic examination lend support to the diagnosis and localization of a brain tumor; the need for more objective or visual evidence makes the performance of one or more of the other tests necessary. All of these diagnostic procedures have certain deficiencies. Ray[8] pointed out some of the shortcomings of air contrast radiography in localizing tumors stating that, "occasionally a normal or apparently normal ventriculogram occurs in the presence of an infiltrating glioma or very small neoplasm (particularly metastatic)." However, he believes this is not a serious deficiency since such lesions are not likely to be benefited by surgery.

With pneumoencephalography or ventriculography very rarely is a tumor observed to be completely surrounded by air, so in these tests the diagnosis and accurate localization of tumors is determined by distortion of the ventricular system, or other alteration in the size, shape and position of the cerebrospinal fluid spaces. Therefore, some tumors may be easily missed because of their size or growth characteristics. Angiography, on the other hand, is of value only if the normal relationships of the cerebral vasculature are altered, or if newly formed vessels within the tumor produce the "tumor blush" on x-ray examination. The limitations of the electroencephalogram seem to vary, particularly with (1) the technic employed, (2) the rapidity of the tumor's growth, and the site of the lesion within the brain.

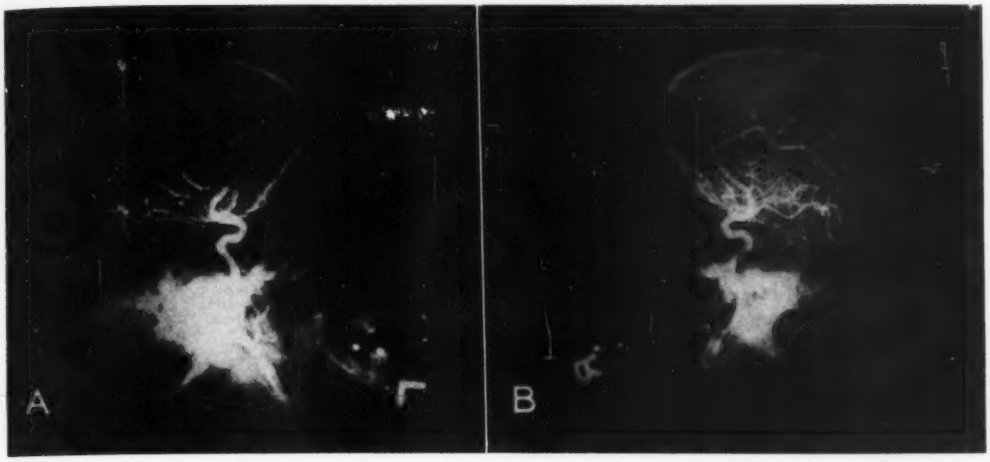
Figures 1 and 2 show portions of the initial

FIG. 1



Shifting abnormal slow wave discharges are noted on preoperative EEG. tracings on September 9. Reported as abnormal and suggestive of "parasagittal tumor or space occupying mass."

FIG. 2



(Same case as in Fig. 1.) Preoperative bilateral carotid arteriograms show good filling of internal cerebral circulation with no displacement on September 10. Interpreted as: "No evidence of expanding intracranial lesion."

angiographic and electroencephalographic studies performed on one of the patients included in the "Diagnostic Problem" group of 40 patients. (Air studies on this patient, pneumoencephalograms, were reported as unsatisfactory on one occasion and normal on another.) This is certainly not typical of the entire group, but this case was chosen to demonstrate the not uncommon phenomenon of a striking abnormality with one procedure and a completely negative result with one or more of the other diagnostic procedures. The case is also atypical in that the development of continuous focal motor seizures, plus the electroencephalographic abnormalities led to surgical intervention in the face of normal and unsatisfactory angiography and air studies.

Percentages of localization related to tumor type and site will not be presented in this paper, but as reported by Kershman, Conde and Gibson[9] the accuracy was greater for the supratentorial masses.

The per cent accuracy of the EEG. in the over-all series is somewhat different from that reported by other investigators; it is thought that this may possibly be accounted for by differences in technic. For example, Cobb[10] in 1944, reported a 50% accuracy, Schlesinger and Strauss[11] in 1947, reported a 43% accuracy, Cuneo and associates[12] in 1950, and Voris[13] in 1952, reported approximately a 48% accuracy. In 1953, Martin, Webster, and Gurdjean[14] reported a 47% accuracy. In some of these reports the technic of recording is not commented upon, but in all instances from one to three variables are noted, as either (1) electrode placement, (2) number of electrodes, and/or (3) monopolar-type of recordings.

Other than the percentage of accuracy obtained, it must be pointed out, however, that the EEG. offers many advantages as a screening procedure. First, lesions involving cortex or subcortical nuclear structures may early show EEG. abnormalities without changes being observed by other technics. (The depressed or irritated aggregates of neurons may give rise to slow waves, or epileptic discharge long before angiographic, or pneumographic alterations are noted.) Second, the ease of administration of the procedure with minimal risk and discomfort is obvious, and the frequency with which it may be used is unlimited. This allows for examination of patients at regular intervals with minimal danger and expense. The procedures described appear to yield reasonable accuracies; the question is to what extent can they be improved, particularly those in which the risks and discomfort to the patient are minimal, the cost low, and the availability universal.

Within the past few years reports have been appearing in the literature regarding

the use of radioactive isotopes in the localization of brain tumors, (Ashkenazy, Davis and Martin[15] and Lassman[16]), both through the intact skull, and after the skull has been opened. Radioactive substances, usually diiodofluorescein, concentrate within the cellular tumor, then the tumor is localized by determining the site of maximum gamma radiation with the aid of various types of scintillators and Geiger counters. The reports indicate a remarkable degree of accuracy, but these substances are quite expensive, are not readily available, and are considered dangerous, particularly for repeated use. With further study some of the shortcomings may be overcome, and if this proves to be so, such substances may offer to this problem even more valuable information through another relatively simple procedure.

Addendum

Since this paper was initially prepared, 19 additional patients having proven brain tumor have been admitted to our hospital for treatment. Their records have been reviewed, and the preoperative diagnostic procedures were evaluated for degree of accuracy in localization in exactly the same manner as the similar procedures performed on the original 99 patients reported in this paper.

A total of 59 preoperative diagnostic procedures were performed on these 19 patients, including 23 angiograms, 23 electroencephalograms, and 13 air studies. After correcting for subsequent examinations, a total of 47 procedures were statistically compared. These include 19 angiograms yielding a 68.4% accuracy, 16 electroencephalograms with an 81.2% accuracy, and 12 air studies with a 58.3% accuracy.

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The Camera in Proctology*

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IN THE PROGRESS OF MEDICINE, the study and dissemination of knowledge of diseases of the anorectum have not kept pace with the advances of other anatomic regions. While there are many factors to explain this lag, i.e., mores, prudery, and esthetics, one of the most important has been the difficulty of accurate communication. To transfer thoughts, too much dependence has been placed upon spoken or written words, which may possess ambiguity and multiplicity of meanings. To more effectively communicate information, the modern convenient medium, the photograph, has been significantly neglected in proctology.

This report is presented to relate some of the benefits experienced in the past seven years with a camera in proctology, to consider some factors that have, perhaps, deterred the use of photography in proctology and, hopefully, to stimulate more interest in the photography of anorectal lesions. Progress in any field of endeavor can be accelerated by improving the media of communication. The physicians of today can promote the pace of progress of proctology by encouraging and practicing the more frequent use of photography in this specialty.

Benefits of the Use of the Camera in Proctology.

During the past seven years, over 7,000 color photographs have been taken of normal and abnormal anorectums. Some pictures have been taken through the proctoscope; most were external views of the anal and perianal areas. Most photographs were taken serially to depict the progress of a lesion since a single photograph constitutes a mere moment of the case. All color slides, taken with Coreco, Leica, or Alpa cameras, have been numbered, mounted, filed, and indexed.

After having been catalogued the slides have served beneficially in the following ways, (1) documenting case records, (2) teaching, (3) research, (4) talks, and (5) publications.

(1) *Case records.* The photographs produced a more accurate record of observations than that permitted by written words alone. The difficulty of choosing proper words to clearly describe a procotologic lesion has been experienced by most clinicians. Proctologic terminology is not the paragon of precision. If the lesion has not been accurately described on the original record, the usefulness of the written record decreases in direct proportion to the inadequacy of the description. The photographs stimulated the memory, recalled and provided a better comparison of the cases than that afforded by written records alone. This continuous self-education by reviewing experiences is essential in the development of a good physician. The photographs of one's cases provide excellent educational material as the physician continually studies the lessons learned in his own practice.

(2) *Teaching.* Visual presentation increases the power of retention and is of established value in education. At such times when clinical material for teaching medical students, nurses, and house staffs was not obtainable, photographs of proctologic lesions have been used. Usually, the number and variety of cases that can be demonstrated to the class by color photographs are much more than those in the hospital or clinic available for teaching purposes.

(3) *Research.* At the Medical Center Hospitals and Clinics, the camera has been used for many years in proctologic investigations.[1,2] The photographs, recording objective evidence to assure validity, have substantiated the reports of many clinical studies dealing with antibiotics and nitrofurazone. The serial color photographs of the cases treated were compared with a similar series from the control group. Photographs improve the quality and scientific worth of the investigation, even though it added to the costs.

(4) *Lectures.* At medical society and staff meetings, photographs provide a more accurate transfer of knowledge than is afforded by words alone. Aside from the feeling of

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pride in the pictures shown at case presentations, photographs permitted a better understanding of the relationship of the pathologic lesion and the anatomic site. They promoted (to use a legal phrase) a "meeting of the minds."

(5) *Publication.* Good pictures are eloquent because they communicate more to the reader than do pages of words. This is especially true in proctology. Pictures often catch the eye before the reader's interest is attracted to the title or to the text. Moreover, pictures are a universal medium of communication.

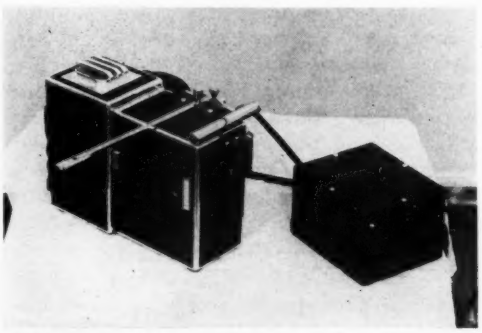
Reasons for Previous Limited Use of Cameras in Proctology.

Because of possible embarrassment, prudery, or inconvenience, some physicians may find that it may not be desirable or feasible to send a patient to a lay photographer. It is, therefore, necessary for the physician to be able to take his own photographs. With the simplicity and technical ease of available modern photographic equipment it is possible for every physician to learn the fundamentals of photography and to act as his own photographer.

There are a large variety of cameras available today for clinical use. Some are manufactured especially for close-up work, others require the addition of a lens, or the addition of an extension tube to the camera to adapt it for close-up photography (Figs. 1-4).

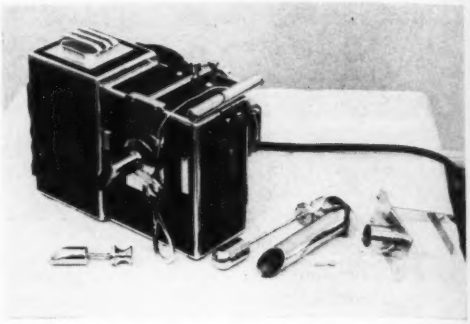
Before purchasing a camera and photographic equipment, the physician should plan in advance what his needs will be. He should decide whether proctoscopic or external lesions or both will be photographed. He should have an idea to what specific or general use he will make of his pictures. This study beforehand will help him to know whether he will need color, or black and white photographs, or both. Whether the physician will use the camera in only the unusual or in all proctologic cases will help him to decide whether to invest in a larger camera using larger and relatively more expensive film for clearer pictures, or a smaller and more adaptable camera for smaller but satisfactory pictures. It is not improbable that the physician might become so absorbed in photography as to assemble and construct equipment to fit his own particular needs. He might even acquire a delightfully absorbing hobby, not an undesirable asset in the midst of his ever-driving vocation.

FIG. 1



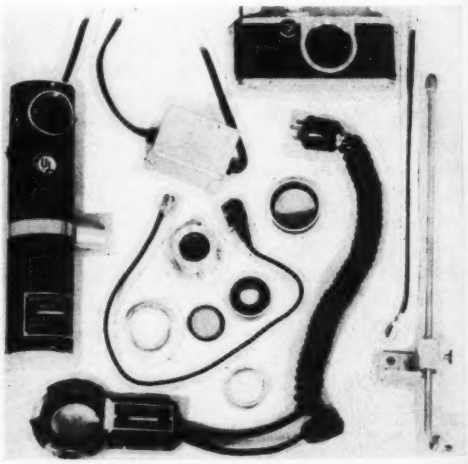
Transformer and electronic flash camera (Coreco) with detachable rod for fixed focus pictures.

FIG. 2



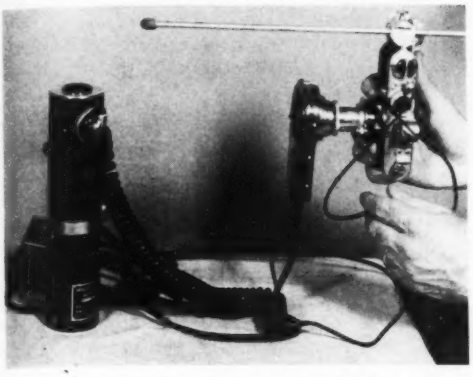
Electronic flash camera (Coreco) with attachments for endoscopic pictures.

FIG. 3



Leica camera, electronic flash circle light, transformer, lens, and attachable device to provide a fixed focus.

FIG. 4



Leica camera and accessories assembled for use.

In this limited report many technical matters in taking satisfactory pictures can not be included. However, some significant points should be mentioned. The camera and accessories should be relatively easy to operate quickly and efficiently. It is desirable to standardize the film, lighting, aperture opening, and distance between camera and object. This uniformity of photographic technic will tend to prevent variations in color and size to decrease errors of misinterpretation.

Quite possibly, because of not being sure of medicolegal considerations, the physician might be reluctant to use a camera in his practice. Actually, the photograph of the anorectal lesion documents a proctologic case record in the same way that a roentgenogram of the chest documents a case record in tuberculosis.

Placing his confidence in the physician, the patient would expect the photographs of his anorectal region to be ethically handled to increase medical knowledge. Moreover, the patient rightfully would resent the frivolous and improper use of the photographs. It is up to the physician not to do anything that might lessen the patient's confidence and trust.

Some cases have been tried in court in which physicians have had to defend claims based upon the theory that there has been an invasion of the right of privacy, or that the plaintiff has been so improperly exposed and exhibited as to have suffered shame and mortification. Just where the patient's right of privacy ends and the public interest begins is a question that can be settled only by specific cases. However, if the physician ever mindful of his responsibility handles the photographs in an ethical manner, he has little to fear of a claim. Regan expressed the problem in the following manner "One is not entitled to have complete isolation under his right of privacy but rather individual rights must be construed in the light of duties incumbent upon individuals as citizens of a free country. At some point the public interest in obtaining information becomes dominant over the individual's desire for privacy."[3]

With the close-up cameras that have been used, photographs have depicted only the anorectal region. Therefore it is extremely unlikely that the identity of any patient can be established.

To avoid any misunderstanding, the patient is told that as a dentist wants pictures of the teeth or an internist requires roentgenograms of the chest, the proctologist needs pictures to provide an accurate record to treat and follow the anorectal lesion. When properly explained that photography is a medium to produce a more accurate record of his anorectal disorder than written words alone, the patient accepts the use of the camera as a routine matter, which it should be.

Whenever the subject of photography in proctology was brought up after operation or treatment, no patient has felt displeased. In fact, many patients expressed a curiosity to view the photographs of their own cases, and they did.

Discussion

In the dissemination of knowledge of diseases of the anorectum, there is a demanding need for improvement in the medium of communication. Words are inadequate to satisfactorily transfer ideas. Language is not static. Moreover, the meanings of words change and people are often unaware of the changes. Pictures convey information better. Moreover, pictures transcend language barriers. When photography is not used, the physician of today is behind the times in not utilizing all the available media of documentation and communication. Because they have not been taught to use a camera to take clinical pictures, many physicians have procrastinated in securing a camera for their practice. To overcome this educational lack, future books on proctology by incorporating

a chapter on photography could emphasize the relative ease of picture taking with modern cameras.

Just as roentgenograms are made to illustrate the course of pulmonary lesions, so photographs should be made to record the progress of proctologic lesions. Furthermore, the photographs would provide permanent objective evidence to document the case records. Also, as a hemoglobinometer is a necessary instrument for the physician to indicate the course in treatment of anemias, so a camera should be considered as necessary equipment for the physician to document the progress of anorectal disorders. The camera should be standard equipment for those who treat diseases of the anorectum.

Progress in proctology can be accelerated by the increased use of the photographs as a more accurate medium of communication. With a photograph a physician can explain with greater clarity and accuracy the anorectal disorder to his patient and to other physicians.

To strive for uniformity of photographic technic, a committee under the auspices of the American Medical Association should be set up to coordinate other interested groups to propose standards. (There is standardization among radiologists as to the size of film, etc.) If all proctologic cameras have a uniform distance from object to lens to film, photographs could be more readily compared and would lessen chances for misinterpretation.

Summary

This report brings into focus some of the difficulties of communication in proctology in the past, and spotlights a way to promote progress in proctology in the future. Today, cameras are available which are technically easy to use so any physician can photograph anorectal lesions. Since words are inadequate to describe an anorectal lesion, photographs improve the clarity and accuracy of the transmission of ideas. By improving the medium of communication, progress in the study and treatment of diseases of the anorectum can be accelerated. To increase the dissemination of accurate information the camera should be standard equipment for the modern physician who treats anorectal diseases.

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The Use of Adjunctive Drugs in the Treatment of Diseases of the Urinary Tract*

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ADJUNCTIVE, by definition, means forming an adjunct, which is something added to another thing but not essentially a part of it. Medically, we refer to adjunctive drugs as those added to a medical regimen to alleviate certain symptoms, as compared to those drugs which are designed to eliminate the basic infection.

Many new drugs have been developed in recent years which were not originally intended for use in the treatment of disease of the urinary tract. Due to their physiologic action they have been adopted by the urologist and play important roles in the alleviation of certain symptoms of disease of the urinary tract. The purpose of this paper is to review some of these adjunctive drugs in the light of my experience with them. Among these to be considered for the purpose of this paper are: (1) the anti-inflammatory-anti-edema group, consisting of streptokinase-streptodornase (Varidase Buccal), chymotrypsin (Chymar Buccal and Chymar injectable); (2) the antispasmodic group, consisting of a combination of hyoscyamine sulphate, atropine sulphate, hyoscine HBr, and phenobarbital (Donnatal), a combination of isopropamide and prochlorperazine (Combid), valethamate Br (Murel), and propantheline Br (Pro-Banthine); and (3) the hormonal group, namely, testosterone (Buccal Linquets).

Our group recently did a study of the anti-inflammatory-anti-edema group in disorders of the urinary tract. Chymar Buccal, Chymar injectable and Varidase Buccal have as their basic function the relief of swelling, inflammation and pain, a well-known triad to the urologist. Chymar contains purified chymotrypsin extracted from mammalian pancreas. Varidase is a purified and concentrated combination of streptokinase and streptodornase (enzymes) produced by a strain of streptococcus.

Generally speaking, this group of drugs is contraindicated when there is evidence of a defect in blood coagulation, such as: (1) afibrinogenemia, (2) in extensive liver damage, (3) acute hemorrhagic pancreatitis, (4) in acute renal insufficiency, and (5) cautious use if there is a history of collagen disease.

Few allergic manifestations have been reported. According to the literature some elevation of temperature may be expected following Varidase. A few patients have shown local irritation and dryness at the site of the disintegration of the tablet, and a few cases of urticaria and rashes have been reported. Mild glossitis has been reported and an occasional mild gastrointestinal disturbance, characterized by moderate fullness or rumbling of brief duration.

Fifty-six cases were studied in our series. The cases chosen were those in which, in my opinion, edema played a major role. Chymar injectable was given primarily to those patients who had nausea and vomiting as part of their illness and the Buccal tablets to those who did not.

The types of cases were as follows: (1) urethral strictures, with or without infection in 7; (2) benign prostatic hypertrophy with infection in 13; (3) acute prostatitis with difficulty on urination in 13; (4) epididymo-orchitis in 13; (5) chronic prostatitis with perineal symptoms in 5; and (6) a miscellaneous group, consisting of:

1. Edema of the corona (insect bite) in 1.
2. Edema of vesical neck after transurethral resection in 1.
3. Edema of testicle and cord after herniorrhaphy in 1.

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4. Hematoma at penoscrotal junction (traumatic) with difficulty voiding in 1.

5. Edema of the ureteral orifice with calculus in 1.

6. Edema of suture line after circumcision in 1.

7. Recurrent urethral syndrome associated with intercourse in 3.

These total to 61 instances, but in several cases two or more conditions were present simultaneously.

The evaluation of results was based on the response of the patient to treatment, as compared to those treated in the past without the use of these adjunctive drugs. It was noted throughout that the best results were obtained in the treatment of acute conditions regardless of diagnosis, and in which treatment was started within the first 24 to 72 hours following the appearance of symptoms. Particularly gratifying were the results in the cases of acute prostatitis and acute epididymo-orchitis. The results obtained in the miscellaneous group were generally excellent with one exception, the one after transurethral resection. Further resection was required in this case. Poor results were obtained in the cases of benign prostatic hypertrophy, and I do not recommend the use of chymotrypsin in these cases. In urethral strictures the results were variable, but there was little doubt that therapy was beneficial in some cases. In chronic prostatitis with perineal symptoms, a few patients were definitely improved though some received no benefit. All instances of recurrent urethral syndrome, associated with intercourse, were benefited.

The allergic manifestations noted were as follows:

With Varidase: (1) One patient experienced a feeling of swelling in the head and ears with mild headache. It was discovered that he swallowed his tablets rather than allowing them to dissolve under the tongue.

(2) One patient developed a small, erythematous area on the buccal mucosa.

With Chymar Buccal: None

With Chymar injectable: (1) One had severe headache, requiring a change to Varidase.

(2) One had an anaphylactoid reaction reported to me by a confrere, Dr. Thomas W. Durham, of Hot Springs.

The conclusion in our study was that these drugs are a relatively safe and valuable addition to our armamentarium. They must be used judiciously and selectively, since certain factors, such as duration of the disease, will certainly influence their effectiveness.

Another drug, which has been most useful in our hands, has been Pro-Banthine. Although this antispasmodic was originally developed for use in gastrointestinal conditions, particularly ulcer, spastic colon and related conditions, it was soon found by the urologists to be of great usefulness in the management of certain urological conditions. Its greatest usefulness in our hands has been in the alleviation of urgency, frequency, and urinary tenesmus in women, who have urethritis, cystitis, or trigonitis. Within 12 to 24 hours there is almost a complete disappearance of these symptoms upon the use of this drug. The patient with the highly irritable bladder who, on original examination, can only hold a few ounces, is so relieved that almost overnight the bladder resumes a normal capacity.

One of the greatest blessings of this drug in our hands has been in the management of bladder spasm after prostatectomy. This is a very distressing condition and may be encountered following any type of prostatectomy, either open or transurethral. It is distressing in that it causes exceedingly great pain, and the spasm itself may definitely promote or prolong postoperative bleeding. Many patients, before we began the use of Pro-Banthine, required excessive amounts of narcotics in high dosage for the relief of this condition. These narcotics definitely impeded the progress of the patient in that he was so continuously "snowed under" that he was unable to maintain a proper diet and fluid intake; second, the majority of these patients has a certain amount of paralytic ileus or an accentuation of any ileus already present. When we first began to use Pro-Banthine, our chief fear was that it would tend to promote ileus. During the course of our early use of this drug we had 2 patients who developed auricular fibrillation. The head of the medical department in our clinic actually believed, for a time, that the Pro-Banthine may have been a contributory fac-

tor; however, we have used this drug now in many cases and believe this was purely coincidental, as we have had no trouble with this item since. Our patients do not receive Pro-Banthine prophylactically. It is used only if bladder spasm actually develops. Thirty milligrams of Pro-Banthine are given intramuscularly in such cases every four (4) hours as needed. Our experience has been that the Pro-Banthine gives these patients quick and prolonged relief, and that usually the bladder spasms disappear altogether within 24 to 48 hours. It has also been our happy experience that the Pro-Banthine has, in no way, promoted or created any ileus. Additionally, there seems to be no residual aftereffect insofar as the patient's ability to void is concerned, because the drug is used for such a short time. Another advantage it offers over the narcotics is that the patients are not depressed and are able to cooperate in the other aspects of their convalescent regimen. I do not hesitate to recommend this drug to you, if you have not used it in your cases in which this distressing symptom occurs.

In recent years the urologist has become more and more aware of the influence of menstruation and the role of sexual intercourse in the production of pelvic congestion and traumatic urethral edema. Many of us are thoroughly convinced that these two factors definitely lead to the development of the female urethral syndrome and other lower urinary tract infections in women. These factors account in many cases for the frequent recurrences. Some of the adjunctive drugs have proven to be very effective in minimizing the symptoms, and even, at times, in actually preventing the recurrences. In the original part of this paper, I pointed out that the use of Varidase may be helpful in many of the female patients with a tendency for recurrence of their symptoms following intercourse. An interesting side light relative to this was a recent communication in the "Urological Letter Club,"[1] submitted by members of the Elmer Hess group in Erie, Pennsylvania. They pointed out that in many of their female patients, who had this tendency for a recurrence of symptoms following intercourse, they had had some successful results using nitrofurantoin (Furadantin). In their program the patient took Furadantin prior to intercourse and again one or two tablets the following day. This is a most interesting observation on their part, but one which I have not followed with any of my patients to date. Working on the same theory as the Hess group, that pelvic congestion and traumatic urethral edema were the trigger mechanisms, we have tried several of these patients on Varidase Buccal tablets. There has been a noticeable reduction in the number of recurrences, since they have been on this program.

For the patients who most frequently develop symptoms at the time of menstruation, we have used Testosterone Buccal Linquets. The dosage has been one daily for 7 days prior to the onset of menstruation. This has greatly reduced the number of recurrences in these cases.

Recently, in the case of a 13 year old girl, this program was instituted and has been carried on now for a period of three months. She has had no recurrences of her urinary tract symptoms under this program. Previously, she had had episodes of urinary urgency, frequency and dysuria over a period of four to five months, always occurring at the time of menstruation and clearing up under routine antibiotic therapy between periods. She had received no antibiotics in this new program and her urine has consistently remained normal. Apparently, this small dosage of testosterone is sufficient to limit the amount of pelvic congestion and edema which occurs and probably provides the physiologic mechanism that helps prevent a recurrence. It is my belief that these drugs can definitely be of great assistance in the management of these chronic and recurrent cases as described.

Summary and Conclusions

A study of 56 selected cases treated with the anti-inflammatory- anti-edema group of drugs—namely, Chymar, Varidase Buccal, and Chymar Buccal—have been presented. It is my conclusion that these drugs have a definite place as adjunctive drugs in the treatment of disease of the urinary tract especially when edema and swelling are prominent factors.

The value of Pro-Banthine has been dis-

cussed especially with reference to bladder spasm following prostatectomy and the management of the irritable bladder.

Testosterone Buccal Linquets have a valuable role in the management of the urethral syndrome in women, especially in those cases where a definite connection seems to exist with either coitus or menstruation.

Acknowledgment

Grateful acknowledgment is made to the Armour Pharmaceutical Company (Chymar Buccal and Chymar injectable) and to Lederle Laboratories (Varidase) for their generous cooperation in supplying the drugs used in this study.

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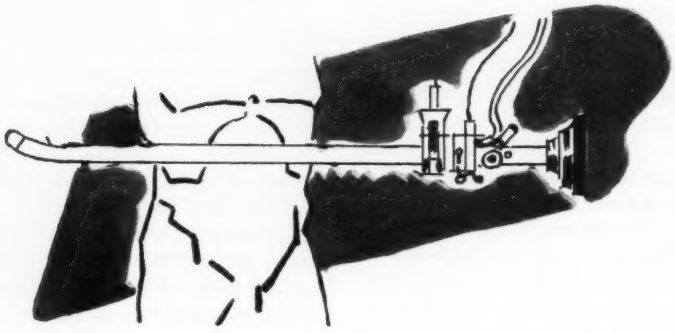

Clinical Training for Public Health*

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When the Advisory Board for Medical Specialities meets in February of 1960 it will have before it a request from the Board of Preventive Medicine and Public Health. This request is for changes in the requirements for education, training, and certification of physicians who wish to devote themselves to the field of Preventive Medicine and Public Health. The plan would offer basic certification in Preventive Medicine, with subspecialty boards in Public Health, Occupational Medicine, and Aviation Medicine to be taken subsequently by those interested in specializing in these fields. The program of training recommended in this proposal requires a year of academic work, with the requirements for two additional years allowing considerable flexibility by providing for a year of clinical residency and a year of research. Clinical knowledge has obvious importance in the basic field of preventive medicine and in the sub-specialties of occupational and aviation medicine, but the question is sometimes raised "What need has a health officer for detailed clinical knowledge? He will not be treating patients." True enough, he may never give direct medical care and yet a firm grounding in clinical medicine seems to me to be essential for the development of public health in the years ahead.

The health officer of 30 years ago held a respected position in the community for he had knowledge and skills necessary for the diagnosis and prevention of infectious and communicable diseases. His clinical knowledge about tuberculosis and other infectious diseases made it possible for him to assume a position of leadership in his community. How can the present-day health officer expect to lead unless he is equally familiar with the basic fundamentals of the problems in which his leadership is now needed?

Many programs need to be developed at the community level. One about which we hear a great deal is a program to combat "chronic disease." This is a broad term including a great many diseases which bear little similarity to each other except that they last a long time. No program can be developed intelligently for any one of these diseases without considerable understanding of the disease process itself and some experience in observing its course and its response to various measures used to treat it or to prevent complications. At our present level of knowledge no over-all community program embracing all chronic diseases can be established except in the area of rehabilitation. To devise an effective program of rehabilitation, put it into action, and give it adequate supervision requires a person who knows something about these diseases, the types of disability they may produce, and the rehabilitative measures appropriate to them. To leave the decisions to someone else is to exercise no leadership whatever. Rehabilitation programs have been instituted at the community level for alcoholics and for patients discharged from mental institutions. A generalized program of rehabilitation has been instituted on a demonstration basis but more of these need to be developed.

Another type of program receiving much attention nowadays is home care. Such programs may be available to patients through a health department, the Visiting Nurse Association, a community visiting nurse service, or a hospital-based home care program. It is possible that insurance companies may soon be offering such benefits through their health insurance programs. The health officer should be ready to assume a role of leadership in such programs. He cannot do so without a sound background in clinical medicine. He may not give the medical services himself but he cannot give adequate supervision unless he knows what constitutes good medical practice. With such knowledge and a home care program serving the physicians of the community he could help materially in raising the standards of care even as he once helped in providing good care for patients with infectious and communicable diseases.

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Many of our current problems need to be met by community action and the public health physician should be leading the way. The aged in our society constitute an ever-increasing problem with manifold ramifications: physical, mental, social, and economic. Leadership in solving these problems can only come from one who has an understanding of all facets of aging. Nursing homes and homes for the aged are rapidly increasing in numbers. Supervision of these homes and establishment of proper standards of care can be done effectively only by one who understands the problems they must face and try to solve. These and many other areas of concern require firm grounding in clinical medicine.

The need for such programs is great. They can increase the availability of medical care while maintaining control of patient management in the hands of the private practitioner. The costs of hospitalization are steadily mounting. A visiting nurse service, a community rehabilitation program, a follow-up program for patients discharged from mental institutions, provision of adequate nursing and rest home facilities all help decrease the cost of medical care by decreasing the length of hospitalization required in a general hospital. At the same time they make such services available to a segment of the population who would otherwise not receive them. Developed locally at the community level, they can be devised to meet local needs and to respect the wishes of local physicians. The need for community medical care programs is becoming more and more pressing; many such programs are being developed to meet these needs. But their value to physicians and patients alike is dependent on adequate supervision by one familiar with the principles and practices of good medical care.

Of equal importance is research. One of our problems in devising adequate community programs is our lack of crucial knowledge concerning the factors resulting in chronic disease and the factors responsible for the disability of aging. Not all elderly people need medical treatment or custodial care. Why do some while others do not? Some chronic diseases are related in their onset to the individual's way of life, it seems from present knowledge, but what are the links in the chain and at what point can an attack be made? What measures can be introduced at a community level to decrease the incidence or the degree of disability resulting from these diseases?

We need not limit ourselves to research in chronic diseases and aging. Much work remains to be done in the genesis of accidents and of injury resulting from accidents before public health programs will be truly effective in reducing their frequency. Air pollution and radiation hazards need much further study. There are administrative problems in giving care and instituting community programs which need attention. And a great deal more time and effort needs to be put into the problem of communication which is essential to health education. A single approach to health education cannot be effective at all social and educational levels and yet it would seem from viewing the activities of some health departments as though it were assumed that this could be done. Many other problems might also be cited. An active research program is a stimulating experience for those engaged in it and for those who come in contact with it. It can revitalize the field of public health.

The people who have framed the new departure in training and certification for a career in Preventive Medicine and Public Health are looking forward. I have outlined a few things that need to be done now. None of us can see what the problems of tomorrow will be. It seems safe to say, however, that the closer the contact of public health workers with clinical medicine and research the more quickly and effectively will they find solutions to these problems as they arise.

Pancreatic Replacement Therapy in Fibrocystic Disease: A Preliminary Report

Concerning A New Pancreatic Extract*

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Anything which may be of assistance in the malabsorption associated with fibrocystic disease is welcome. The authors report their experiments with a new pancreatic extract.

FIBROCYSTIC DISEASE is a hereditary disorder manifested in infants and children by a dysfunction of almost all exocrine glands. Individual organs may be affected to varying degrees, but the fully developed syndrome consists of pulmonary disease, pancreatic insufficiency resulting in steatorrhea, and the excretion of high concentrations of chloride and sodium in the sweat. The mucous glands of the tracheobronchial mucosa secrete an abnormally viscid mucus which obstructs the smaller air passages and causes chronic infection, fibrosis, and emphysema. Pancreatic secretion is also abnormally viscid. The pancreatic ducts become obstructed and are at least partially responsible for the pancreatic insufficiency.

Therapy for fibrocystic disease has been directed primarily toward prevention of recurrent pulmonary infections and improving the nutritional status of the patient by using pancreatic extracts. Many pancreatic extracts available today are deficient in lipase activity. For this reason, large amounts (20 to 40 Gm.) may be required daily to control steatorrhea due to pancreatic insufficiency. The deficiency of lipase activity in pancreatic extracts has been due chiefly to the lack of a reliable assay of the extracts.[1] Recently, a new pancreatic extract, potent in lipase activity, was made available to us. This extract, known as "Pancreatic enzymes, concentrated" (P.E.C.)* is prepared from hog pancreas. A method, based upon the liberation of glycerol from fat hydrolysis, has been devised to assay the potency of lipase activity in the extract. It has been determined that one unit of the lipase activity will hydrolyze about 8 mg. of fat.[1] The extract is supplied at present in small capsules (approximately 100 mg. each), containing 1,000 units of lipase activity, as well as adequate trypsin and amylase.

This report is (1) our preliminary experience with the effect of this pancreatic extract on the digestion and subsequent absorption of radioactive labeled neutral fat, and (2) the use of this preparation in the management of pancreatic insufficiency in patients with fibrocystic disease.

Materials and Methods

Thirteen children with fibrocystic disease were studied. This group consisted of 7 boys and 6 girls whose ages ranged from 3 to 13 years. The diagnosis was established in each child by history, clinical manifestations, demonstration of pancreatic deficiency, and by use of the sweat test.[2]

Initially, each child was hospitalized for a period of 6 to 8 days for observation and for conducting fat absorption tests using I^{131} triolein (radioiodinated glyceryl trioleate). Each child was given a test meal containing 15 to 20 μc of I^{131} triolein, and venous blood samples were collected at 1 to 2 hour intervals for 6 to 8 hours. All feces passed during the next 48 to 72 hours were collected. Radioactivity in the blood and fecal samples was determined by measuring the radioactivity in the specimens with a well-type scintillation counter

*Read before the Section on Gastroenterology, Southern Medical Association, Fifty-Third Annual Meeting, Atlanta, Ga., November 16-19, 1959.

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*The pancreatic extract used in this study was kindly furnished to us by Mr. Kenneth Ericson, Organon Inc., West Orange, New Jersey.

and expressing the results as a percentage of the administered dose. The methods used were similiar to those recently described by Isley and associates[3] and Owen.[4] After all radioactivity had disappeared from the stool following the initial test, the entire procedure was repeated with 300 mg. (3,000 lipase units) of the pancreatic extract added to the test meal.

Patients with moderate or severe pancreatic insufficiency, as indicated by the results of the fat absorption test, received replacement therapy consisting of three capsules of the pancreatic extract (300 mg.) with each meal. The parents were requested to keep a record of the number of stools passed by the child each day, to weigh the child once a week, and to measure his height once a month. Following replacement therapy for from 3 to 5 months, 6 of the children have been re-evaluated. Fat absorption studies were repeated and the results were compared with the initial test.

Results of the Fat Absorption Studies

In our laboratory, normal values for fat absorption are above 10% of the administered dose of I^{131} triolein appearing in the total blood volume within six hours, and less than 5% of the administered dose excreted in the stool in 48 to 72 hours. All but 1 of the 13 children showed evidence of pancreatic insufficiency as determined by this test. The patient who exhibited normal pancreatic function had moderately severe pulmonary disease, and results of the sweat test were positive. A control test, without the enzyme, was done initially in 9 of the remaining 12 patients to determine the peak concentration of radioactivity in the blood and the amount of radioactivity excreted in the stool. The results are given in table 1.

Table 1 shows that all but 1 of the 9 patients who had control tests had moderate or severe pancreatic insufficiency. The average peak concentration of radioactivity in the blood was only 2.4% of the administered dose. The average amount of radioactivity excreted in the stool was 64.3% of the administered dose.

Three patients were tested initially with commercially available pancreatic extracts. One was given Viokase, 300 mg.; another, Viokase, 760 mg., and another, Pancreatin, 1,000 mg. In none of the children did the peak concentration of radioactivity in the blood exceed 3.8% of the administered dose, and the amount of radioactivity excreted in the stool varied from 19.0 to 27.5% of the administered dose.

When each of the 12 patients was retested with the new pancreatic extract, 300 mg., added to the test meal, improvement in fat absorption, as indicated by the amount of radioactivity appearing in the blood and in the stool, was marked. The average peak concentration of radioactivity in the blood increased to 7.9%, and the average amount of radioactivity excreted in the stool decreased to 14.4% of the administered dose. The results of the second test are given in table 2.

Although improvement was marked, table 2 shows that results of the second test for fat absorption, following the addition of pancreatic extract to the test meal, were not always in the normal range. Improvement of fat ab-

TABLE 1

CONTROL STUDY—WITHOUT ENZYME

Patient	*Sex*	*Age*	*Radioactivity (% of Dose)* *Blood**	*Stool#*
1	M	10	0.7	73.7
2	F	7	5.8	33.4
3	F	8	1.3	49.4
4	M	5	1.0	34.6
5	M	3	0.7	96.7
6	M	5	10.0	9.2
7	F	5	0.5	98.9
8	F	13	0.8	86.5
9	M	4	0.5	95.9
	Mean Values		2.4	64.3

*Peak blood concentration
#Total stool concentration

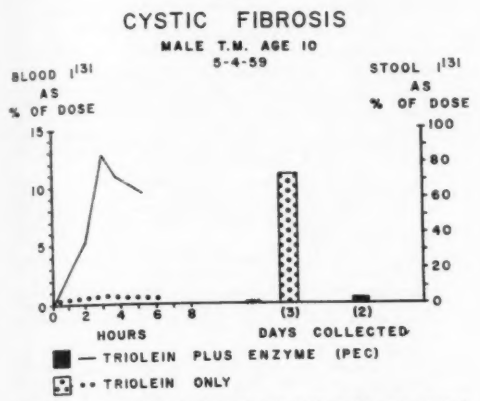
TABLE 2

ENZYME STUDY—300 MG. P.E.C.

Patient	*Sex*	*Age*	*Radioactivity (% of Dose)* *Blood**	*Stool#*
1	M	10	12.8	4.3
2	F	7	14.3	6.0
3	F	8	5.5	17.8
4	M	5	5.5	6.6
5	M	3	7.0	6.3
6	M	5	9.6	3.0
7	F	5	2.1	42.3
8	F	13	7.3	27.8
9	M	4	12.3	9.5
10	M	5	2.7	13.8
11	F	4	6.9	7.1
12	F	8	8.3	27.8
	Mean Values		7.9	14.4

*Peak blood concentration
#Total stool concentration

FIG. 1

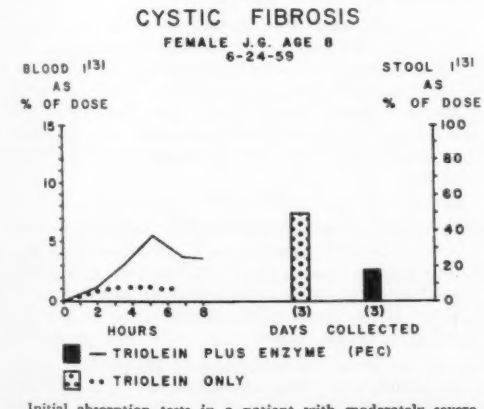


The control test for fat absorption is indicated by the dotted line and the dotted bar. On the left are the curves of radioactivity in the blood before and after the addition of 300 mg. of pancreatic extract (P.E.C.) to the test meal. The bar graphs on the right indicate the excretion of radioactivity in the stool specimens. This patient had 4 to 6 foul, bulky stools daily and had not been on previous pancreatic replacement therapy.

sorption in one of the patients, tested first without and then with the new pancreatic extract added, is illustrated graphically in figure 1. After maintenance therapy with the pancreatic extract for 2 to 3 months, testing indicated additional improvement in fat absorption in some of the patients (Figs. 2 and 3).

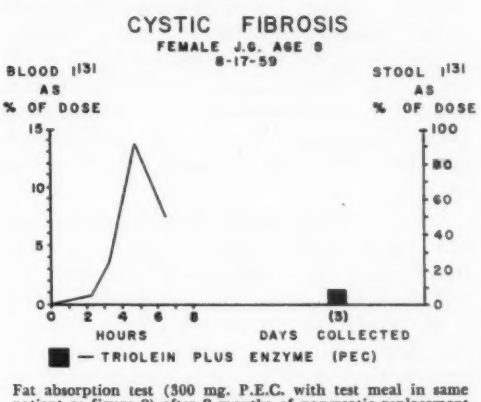
Other pancreatic extracts were added when some of the patients were tested. To provide a comparison of the value of different pancreatic extracts in the same patient, fat absorption was evaluated following ingestion of each (Fig. 4).

FIG. 2



Initial absorption tests in a patient with moderately severe fibrocystic disease. The control test is indicated by the dotted line and dotted bar graph. When 300 mg. of P.E.C. was added to the test meal (solid line and bar) the radioactivity appearing in the blood and stool specimens showed improvement in absorption, but normal values were not attained.

FIG. 3



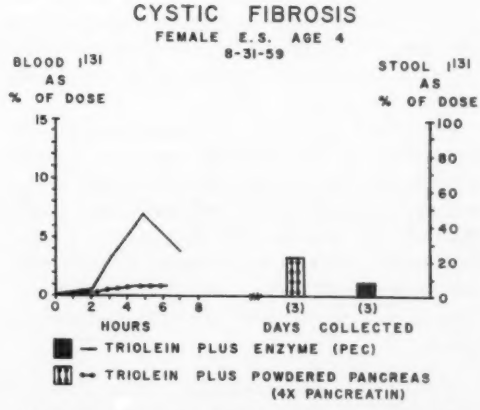
Fat absorption test (300 mg. P.E.C. with test meal in same patient as figure 2) after 2 months of pancreatic replacement therapy with P.E.C. Note that the curve of radioactivity in the blood is now normal, and the amount of radioactivity excreted in the stool is only about 50% of that found when 300 mg. of P.E.C. was added to test meal on June 24, 1959.

Clinical Evaluation

A program of replacement therapy was instituted in each child demonstrating moderate or severe pancreatic insufficiency. All visible fats were eliminated from the diet and 300 mg. of the pancreatic extract was added to each meal. Supplementary vitamins were continued as well as antibiotics and the other medications used in the treatment of the pulmonary complications.

In each of the patients placed on maintenance therapy, an immediate decrease was noted in the number of stools per day. Although many of the patients' appetites de-

FIG. 4



Comparison of two fat absorption tests in a patient when 300 mg. of P.E.C. was given with test meal (solid line and bar) and when 760 mg. of Viokase was added to test meal.

creased, all have shown increases in weight, physical activity, and well-being. Intolerance to the preparation such as nausea, vomiting, or abdominal discomfort has not been noted. Because a relatively small quantity of the pancreatic extract is necessary to control the symptoms of pancreatic insufficiency, the preparation has been well accepted by all of the patients.

An interesting observation has been that many of the children were chronic bed-wetters, and the problem of enuresis became much less or disappeared completely after administration of the pancreatic extract for a few weeks. Although experience has been too limited to determine the long-term effect of pancreatic replacement therapy with this preparation, preliminary observations are encouraging.

Comment

The use of I^{131} triolein as a test of pancreatic function is well established. The appearance of radioactivity in the blood and the excretion of radioactivity in the stools are indications of fat absorption.[5,6] The I^{131} triolein absorption test has demonstrated that most patients with cystic fibrosis have marked pancreatic insufficiency.

The fact that the units of lipase activity in this pancreatic extract can be expressed in terms of the quantity of fat hydrolyzed is of definite advantage. It allows one to calculate the dose of pancreatic extract needed to digest the amount of fat contained in a meal. For example, if a meal contains 24 Gm. of neutral fat, an amount of pancreatic extract containing 3,000 units of lipase activity would be needed for the complete digestion of the fat in the meal. This would be three of the small capsules of pancreatic extract, each containing 1,000 units of lipase activity.

When 300 mg. of the new pancreatic extract are added to the patients' regimen, the I^{131} triolein test reveals that fat absorption either becomes normal or is markedly improved. During the few months that patients have been maintained on replacement therapy with this preparation, clinical observations indicate that it is a potent and effective therapeutic agent.

Acknowledgment

The authors wish to acknowledge the cooperation of Dr. Paul L. Richburg, Dallas, Tex., who kindly referred several of these patients to us for the fat absorption studies.

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Discussion (Abstract)

Dr. W. Crockett Chears, Jr., Dallas, Tex. The data presented by Dr. Best and his co-workers represent in effect a bio-assay of a new preparation of pancreatic extract by means of the observation which, in my opinion, is the most reliable *in vivo* measure of pancreatic lipase activity, that is, the determination of the amount of radioactivity excreted in the feces after the oral administration of radioactively labeled fat.

The results are a convincing demonstration of the potency of the material. One would anticipate, as the follow-up on these patients is beginning to show, that effective pancreatic enzyme replacement and substantial nutritional benefit can be obtained through its use, apparently more so than with other products now available.

Most interesting is the observation that the fecal values in these patients are nearly normal when the preparation is given with the tagged test meal before institution of regular pancreatic extract therapy but that the blood values become normal in some of them only after a period of such therapy.

Little data have been presented as to the reproducibility of the results with this technic of either the blood or the fecal values in abnormals, and one must be cautious in drawing a conclusion. Since the fecal values are similar before and after the period of pancreatin therapy, one would conclude that the overall assimiliation of fat was about the same on both occasions.

Among the hypotheses which suggest themselves in explanation of the difference in the blood values are:

1. More rapid fat utilization in the chronically fat starved patient. Against this is the observation (Chears, unpublished data) that after tagged fat meals given to patients on *low fat diets,* the blood values tend to be significantly higher rather than lower than normal.

2. It has been reported recently (Reemtsma, Pediatrics 22:525, 1958) that absorption of *fatty acid* (as revealed by the blood curves of radioactivity after a radioactively tagged fatty acid meal) was abnormal in each of eight patients with cystic fibrosis manifested by low tryptic activity of duodenal fluid. Fecal values

were not obtained in this study. This finding suggested that there may well be a non-pancreatic factor in the steatorrhea of cystic fibrosis. It may be that this is a reversible defect, perhaps related to malnutrition, possibly true intestinal malabsorption or possibly a "disuse atrophy" of the intestinal epithelial fat mobilization mechanism, since Dr. Best's data suggest rather complete absorption in the presence of a potent source of lipase. This observation certainly warrants further study.

Last, I should like to compliment Dr. Best and his co-workers on their fine work and on their industry. Blood curves and complete fecal collections repeated several times on even a small number of patients represent a great deal of work and a valuable contribution.

ST. LOUIS

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Spondylolisthesis and Spondylolysis:

A Clinicostatistical Report on 589 Cases Treated Over a Thirteen Year Period (1945 Through 1958)*

ELIAS MARGO, M.D.,† *Oklahoma City, Okla.*

The congenital nature of these lesions is discussed, as well as the modes of treatment. The author has had good results in the majority of patients treated surgically; though conservative treatment is always given a trial before operation.

SPONDYLOLISTHESIS is an uncommon condition, yet it occurs often enough to be of practical importance in the differential diagnosis and treatment of backache. It has been recognized as a forward displacement of the body of the fifth lumbar vertebra on the first sacral vertebra, less frequently at other levels in the spine.

Spondylolisthesis is a defect in the spine at the junction between the articular processes and pedicles, with separation of the body and pedicle from the posterior elements of the vertebra. The body and pedicles of the affected vertebra tend to slip forward while the laminae and spinous processes remain with the vertebra below. The defect is congenital and occasionally is unilateral. Displacement may be negligible or marked. The fifth lumbar vertebra is most commonly affected. In some instances the fifth lumbar vertebra slips completely forward over the anterior lip of the sacrum. The defect in spondylolisthesis may be narrow and filled with firm fibrous connective tissue or it may be wider and filled with loose fibrous connective tissue.[1]

Reverse spondylolisthesis is the opposite of the condition described above. Infrequently the displacement may occur in more than one intervertebral space, hence the term "double spondylolisthesis." Also, spondylolysis may occur, with or without displacement.[2]

Pain usually begins gradually though it may begin suddenly. The patient frequently is seen on the day of onset of pain, or at the time of trauma. More often it is days, weeks or months after the onset of pain before the patient is seen. The pain may be dull or sharp, may be present continuously or only with activity or on certain motions or actions. There may have been a definite injury such as a fall or heavy lifting at the onset, or the pain may have begun gradually and without apparent cause. The pain is usually relieved by rest, heat or a support. Weakness and fatigue are very common but may not be outstanding. Stiffness is less common but is sometimes marked. Disability is usually present and varies with the amount of activity and the severity of symptoms. Tenderness at the lumbosacral angle, and pain to deep pressure or percussion over the spine of the fifth lumbar vertebra are usual, and spasm of the erector spinae muscle is common. Hyperextension of the hips is rarely limited. Straight leg raising is often limited on one side, especially when sciatica is present. The lumbar motions may be limited and painful in any, or all directions.

The normal anatomic peculiarities and function of the lumbosacral joint in man naturally predisposes to subluxation.[2] The articulating surfaces of this joint slope at a downward angle of 30 degrees; the superior articulating surface of the sacrum is beveled anteriorly, and there is normally a greater strain on this joint than any other in the spine because of the superincumbent weight which reaches a maximum at this point. Subluxation of the whole fifth lumbar vertebra may occur in the absence of congenital defects or variations. Bad postural habits or previous lesions of the back, such as sacroiliac strain, cause the ligaments to weaken

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and stretch, allowing the articular process to give and widen on extreme forward bending.

Many authors have shown that the lumbosacral joint is the site of the greatest number of congenital defects and variations in the vertebral column.[3] Since the ossification of the vertebrae and closure of the neural canal occur last in this region, often with either failure or delay in closure, it leaves wide defects. In the gradual change of man to the upright position, the lumbosacral region required the greatest alteration to accommodate itself to the new line of weight bearing.

A common defect is the bifid neural arch or nonfusion of the lamina in the midline posteriorly with an absence of the spinous process, leaving no place of attachment for the interspinous and supraspinous ligaments. This allows mobility and predisposes subluxation. In extensive defects of this kind bony anchorage between the vertebrae is lost. Two types of subluxation are encountered;—that of the body with a wide defect or space, and that of the whole vertebra without widening or separation.

Why does spondylolysis develop in the fifth lumbar vertebra in the great majority of cases? "It would seem that this happens because of the special position and relationship of this vertebra in the lumbar curvature of the vertebral column to the position of the sacrum. The fifth lumbar vertebra normally occupies an intermediately oblique position between the obliquely lying sacrum and the almost vertically situated fourth lumbar vertebra. The compressive forces of the spinal column in the higher regions are transmitted for the most part through the neural arches."[3]

In typical spondylolysis, generally found in the lower lumbar vertebrae, the clefts of the isthmus are always located between the downward projecting inferior articular processes of the vertebra above and the upward projecting superior articular processes of the vertebra below. Such clefts appear to be produced by compression of the isthmuses by these articular processes in a mechanism termed as a "pincers effect." The presence of preceding bony abnormalities or congenital defects of the isthmus would seem to be unnecessary for the production of lesions. The anatomic factors promoting the development of this condition in some individuals and those preventing its appearance in others are not clear. The participation of other factors, such as erect posture, trauma, age and sex is to be considered.[3]

Clinical Studies

The chief complaint in the 589 cases (Table 1) we have reviewed in this series has been "backache." This was usually a dull aching pain, often relieved by the conservative treatment of rest and other simple measures, only to recur and worsen with trauma or prolonged activity. Weakness of the back was generally present. Very few of our patients were aware of the deformities present in their spine (Table 2).

All of the patients, with the exclusion of 69, had adequate treatment and form the basis for this study. The 69 patients excluded were seen only for evaluation of disability or for diagnosis (Table 3). Our treatment was conservative or operative. Conservative management was by exercises, back support or both. Surgical correction was done by either laminectomy, by the Gill technic, or fusion (Table 4).

By these methods satisfactory results were obtained in all but 14 instances, which we classified as poor. Six of the 14 patients were treated conservatively and 8 were surgical. Of

TABLE 1

Cases reviewed in this series		=	589
Males	= 465 or 78.9%		
Females	= 124 or 21.1%		
Cases treated by surgery	= 153 or 20.6%		
Cases treated conservatively	= 436 or 79.4%		
Cases having disk disease and spondylolisthesis	= 39 or 6.6%		
	Total	=	589

TABLE 2

Condition Brought to Light By:		
1. Trauma		322
Direct (Due to fall, being struck by object or auto accident)	= 182 or 30.8%	
Indirect (Lifting, stooping or bending)	= 140 or 38.3%	
	322 or 59.1%	
2. Gradual or sudden onset		267
(Etiology Unknown)	= 267 or 40.9%	
	Total =	589

TABLE 3

Private patients	= 381 or 69.4%
Liability cases	= 30 or 4.2%
Industrial cases	= 179 or 26.4%
	589
Housewives	= 109 or 10.7%
Farmers	= 47 or 6.2%
Students	= 40 or 5.8%
Employed in industry	= 393 or 77.3%
Oldest patient treated	85 years of age
Youngest patient treated	7 years of age
Oldest patient treated surgically	72 years of age
Youngest patient treated surgically	7 years of age

TABLE 4

Method of Conservative Treatment	
Application of brace or support	128
Exercises and observation	308
	436
Types of Operation	
1. Rectangular facet bone block grafts	69
2. Circular facet bone block grafts	18
3. "H" type bone grafts	4
4. Interbody bone grafts	6
5. Hibb's type fusion	6
6. Hibb's and onlay bone grafts	14
7. Gill laminectomy	36
	153

the surgical cases only 2 did not admit satisfactory results. In these cases the desire for sick benefits may have influenced the mental attitude of the patient.

Our results were evaluated as *good, fair* or *poor*. Since the patient's own statement of his condition was used as a criterion for this evaluation, all patients having an adequate follow-up of one year or more were questioned as to whether they had symptoms of pain or discomfort and if they had been able to return to their former occupation or activities. They were then evaluated as:

Good—asymptomatic and able to return to their former occupation or activities.

Fair—symptomatic but able to return to their former occupation or activities.

Poor—having pain and unable to return to their former occupation or activities. See tables 5-7.

Since 209, or 30.6%, of our cases were industrial or liability cases the evaluation given was from a minimum of 5% to a maximum of 60% to the body as a whole (Table 8).

Different methods of grading displacement in spondylolisthesis have been described. The method of Meyerding[4] is perhaps the most appropriate and is the method used in this study (Tables 9 and 10).

The importance of early surgical intervention in youth is stressed. To quote from Gill, Manning and White,[5] "Early surgical intervention in the teenager with congenital spondylolisthesis is indicated since these patients are very susceptible to hyperextension injuries. The defect in the pars interarticularis of the vertebra permits progressive forward slipping of the vertebral body, however,

TABLE 5

RESULTS OF TREATMENT

Surgical	
Good	137 or 90.9%
Fair	7 or 4.0%
Poor	8 or 4.5%
Expired under anesthetic	1 or 0.6%
Conservative	
Good	349 or 81.7%
Fair	12 or 3.1%
Poor	6 or 1.0%
Seen or diagnosis only	42 or 8.1%
(Returned to family physician with recommendations for treatment)	
Seen for disability evaluation	27 or 6.1%

TABLE 6

COMPLICATIONS

Postoperative infections	3
Cases requiring revision before relief was obtained	5
Cases with postoperative fracture through fused area	2
Cases having compression fracture of one or more vertebrae	20

TABLE 7

OTHER COMPLICATIONS

Syphilis	5
Arthritis	65
Diabetes mellitus	1
Severe kidney infection	1
Epilepsy	1
Postoperative jaundice (Secondary to transfusion)	1
Postoperative phlebitis in lower extremity	3

TABLE 8

INDUSTRIAL AND LIABILITY CASE AVERAGE

Greatest amount of disability	= 60% To body
Smallest amount of disability	= 5% To body
Greatest industrial time loss (U.S. Compensation case: Would not admit enough relief to return to work)	= 7 years
Shortest industrial time loss (Following Gill procedure)	= 15 days

TABLE 9

GRADE OF DEFORMITY AND TYPE OF TREATMENT

	Grade 1	*Grade 2*	*Grade 3*	*Grade 4*	*Total*
Spondylolisthesis					
Conservative	229	67	6	6	315
Surgical	89	30	7	2	128
					443
Spondylolisthesis with spondylolysis					
Conservative	84	8	1	0	93
Surgical	5	0	0	0	5
					98
Reverse spondylolisthesis					
Conservative	10	0	0	0	10
Surgical	5	0	0	0	5
					15
Double (2 levels) spondylolisthesis					
Conservative	8	0	0	0	8
Surgical	2	1	0	0	3
					11
				Grand total	= 589

if the loose lamina is not very mobile there may be no irritation to the dura for years. If the defect in the pars interarticularis is more posteriorly situated then pressure on the fifth lumbar root could not develop. But, on the other hand, if the defect is anterior then there is likelihood of compression with only minor injury. This pathologic picture explains why many patients go for years without symptoms, since considerable time may be necessary for growth of a fibrocartilaginous mass large enough to compress and irritate the nerve root. This also explains the sudden development of symptoms in these patients following flexion injuries since the fibrocartilaginous mass is then pulled upon adhesions between it and the nerve roots. The jamming down of the loose lamina causes local irritation of the dura with stretching of the first sacral roots and more downward pressure on the fifth lumbar roots which are already in a precarious situation."

I also believe that heredity is a factor in congenital abnormalities of the low back region. As an example a family group of 3 is presented, e.g., the father and two sons.

Case Report—Family Group

Case 1. T. W. A., age 35 years, was admitted on April 14, 1948, with the diagnosis: congenital bifid

TABLE 10

LEVELS OF THE LESION

				Double Levels		
	Lumbosacral	*L4-L5*	*L2-L3*	*L4/5+L5*	*L3/4+L4/5*	*Total*
Spondylolisthesis						
Surgical	115	20	1	0	0	136
Conservative	349	56	0	0	0	405
						541
Reverse spondylolisthesis						
Surgical	10	0	0	0	0	10
Conservative	2	3	0	0	0	5
						15
Double (2 levels) spondylolisthesis						
Surgical	0	0	0	3	0	3
Conservative	0	0	0	8	0	8
						11
Spondylolysis at lumbosacral joint						
Surgical	9	0	0	0	0	9
Conservative	13	0	0	0	0	13
						22
					Grand total	= 589

L-5 to S-1; narrow space, lordosis, asymmetrical facets. There was no known injury. The onset of back pain was insidious with intermittent episodes of dull aching in the hips, low back and left leg. The general physical examination was negative with the exception of tenderness in the lumbosacral area, lordosis and restricted motion. X-ray studies of the spine showed a defect in the isthmus of L-5, increased angulation and decreased lumbosacral joint. Conservative treatment by exercises and support were used for one year. At operation on June 7, 1949, facet bone block fusion to L4/5 and lumbosacral joints was done. The result was *good*.

Case 2. D. A., age 13 years, had an admitting diagnosis of: grade 1 spondylolisthesis, lumbosacral. There was no known injury. The spine had been painful for one year on activity. General examination was normal except for tenderness over the lumbosacral spine and lordosis. X-ray examination showed a grade 1 spondylolisthesis. On May 21, 1952, a facet bone block fusion to L-4 and 5 and lumbosacral joints, plus lumbar fasciotomy was done. The result was *good*.

Case 3. L. W. A., age 15 years, was admitted with a diagnosis of: grade 1 spondylolisthesis, lumbosacral. There was no known injury. The lumbosacral spine was painful. General examination was normal with the exception of tenderness over the lumbosacral joint. X-ray studies showed a grade 1 spondylolisthesis. An operation on June 2, 1952 for a facet bone block fusion to L-4 and 5 and lumbosacral joints, plus lumbar fasciotomy, was done. The result was *good*.

Summary

1. Conservative measures should always be given first consideration in the treatment of spondylolisthesis.

2. Operative treatment, I believe, is indicated in the following:

(a) In youthful individuals in whom the symptoms have persisted in spite of conservative treatment.

(b) In patients with recurring radicular symptoms which defy conservative measures, particularly in the laboring group of persons.

(c) Operative fusion seems to be the logical treatment in severe cases with nerve root irritation or with neurologic findings.

(d) Lumbar fasciotomy aids in reducing the increased lordosis and improves the weight bearing.

3. A total of 39 of our patients had involvement of the disk which necessitated its removal.

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Discussion (Abstract)

Dr. Richard M. Logue, Little Rock, Ark. First, I would like to thank Dr. Margo for the opportunity to discuss his paper. I was furnished with a copy of his very complete and definitive study of this condition two weeks ago. Because of the pressure of time, it was impossible for Dr. Margo to read his paper at the actual presentation and much of the value of the paper has been lost because only the tables accompanying the paper were shown. The classification after the method of Meyerding was carried out in a review of these cases and this was not clearly delineated in the actual presentation because of the lack of time.

We have been impressed with the number of cases seen by Dr. Margo and his group. Certainly in our practice no such series can be evaluated, but I believe in my practice less than 20% have come to operation. I agree with the author that early treatment by surgery is indicated in the youthful individual. I also agree there is some evidence that a congenital factor is present and I have seen one family of three men of advanced age, two of whom have had the Gibbs' procedure. One of these patients is 65, another 68 and another 71. The youngest patient that was seen in our office is a child of 2½ years with a second degree forward slipping of L-5.

I believe Dr. Margo is correct in stating that the Gibbs' procedure is of most benefit in the older patient and those doing relatively sedentary work. I think the exact type of surgical procedure can best be determined at the operating table when the degree of instability of the posterior elements is seen. If they tilt and gig forward into the dura, it would seem that fusing a back without removing or correcting this pressure from the posterior elements might actually "fix" these patients in the position of optimum irritation. Thus, I believe, fusion is best done in the flexed position. With further refinement of the anterolateral fusion, it is thought that eventually the removal of the posterior elements, together with such a fusion might be the best answer to this very real problem.

Again, I would like to say that I enjoyed reading this paper and believe that the group here will be able to derive more from the published material than has been obtained from the slides which were shown today. I would like to ask Dr. Margo if he explores every one of these cases for a disk. I would also like to ask if he routinely takes oblique views to better show the pars-inarticularis defect.

Dr. Margo (Closing). In reply to the discussant's questions my answers are that all cases are explored for nerve root pressure, making a complete examination of the roots and their respective canals. Secondly, oblique views are made only when study indicates disk involvement. Routinely they are not made.

Laryngeal Strictures*

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Persons suffering from this lesion are not doomed to continued disability. There are reconstructive operations which may be applied successfully.

THROUGHOUT THE MEDICAL LITERATURE, the problem of treating laryngeal stenosis has been considered a very difficult one. Although the principles of treatment have changed little during the past 50 years, technics have been refined, and the acrylic and polyethylene plastics have been made available for the construction of stents, so that now we can attack the problem with more confidence and hope for a reasonably speedy rehabilitation of the patient. Still, the following statement made by John Winslow[1] in 1909 holds true: "Many of these cases are excessively rebellious to treatment, requiring patience, persistence, self-sacrifice and skill on the part of the surgeon and endurance of discomfort or even suffering by the patient. Despite these drawbacks, of such seriousness is the condition, and so imperative the necessity for its amelioration, that we are well compensated for making any sacrifice of time and energy that may be necessary for a successful outcome."

Etiology

In the past, most of the strictures encountered were caused by high tracheotomies and complications of infections such as diphtheria, tuberculosis, syphilis, and typhoid fever. Jackson[2] states that 85% of the strictures that they have treated have been caused by high tracheotomies. High tracheotomies are becoming a thing of the past, however, and this factor is not as important as it has been. The automobile is replacing the germ as a frequent cause of laryngeal strictures, and trauma seems to head the list not only in frequency of cases, but in the severity of the problem produced. Other causes include neoplasm, radiation necrosis, congenital defects, corrosive burns, pemphigus and many other conditions that are capable of producing scar tissue or destroying cartilage.

Pathology

To understand the problems one must remember that the lumen of the larynx and the trachea depend upon the integrity of the cartilaginous framework. In fractures of the cartilages there may be overlapping of the fragments and thus a narrowing of the lumen, or portions of the cartilages may be destroyed by radiation necrosis, surgery, or infection and a stenosis be produced. The lumen may also be encroached upon by contractions of cicatricial tissue or by adhesive webs. Strictures may vary in length from a thin web to a solid mass of scar tissue. Occasionally, in the traumatic cases, there will be a bilateral abductor paralysis of the vocal cords to complicate the picture.

Diagnosis

To plan treatment efficiently, one should determine as accurately as possible the site of stricture, the extent and length of it, and whether it is complicated by loss of cartilage or a bilateral abductor paralysis. Examination by mirror palpation and radiographs will usually give enough information. Other means of demonstrating lesions are direct laryngoscopy, tomography, and retrograde inspection through the tracheostomy. A slight stricture may be compatible with normal activity and may cause only noisy breathing on exertion and with little dyspnea. Treatment will probably not improve this situation. However, doubt as to the need for treatment rarely arises, because usually the disability is severe and very definite. Most patients who present themselves for definitive treatment have already become dependent on a tracheotomy and many, even in this enlightened age, have been led to believe they will never be rid of the tracheostomy.

Treatment

A great percentage of post-traumatic strictures might have been prevented by proper early treatment. Any patient who has suffi-

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cient trauma to his larynx to cause endolaryngeal hematomas, edema, and obstruction probably has fractures of the cartilages. Of course, the immediate problem of obstruction is handled by a tracheotomy. When the condition of the patient permits, preferably within the first four or five days, a thyrotomy should be done and the larynx explored. If cartilage fragments can be manipulated into place this should be tried. A plastic mold may be inserted into the lumen of the larynx and left in place for about six weeks. If chronic stenosis can be prevented by this procedure, the result will be much better than that obtained by corrective operation later.

Attempts at overcoming chronic stenosis of the larynx probably began with Von Schwetter,[3] of Vienna, in 1871, when he devised methods of dilatation using metal olives when a tracheotomy was done and rubber tubes inserted preorally when a tracheostomy was not present. Open operations were also done in these early days, and a method of treatment involving a laryngotracheostomy with dilatation under direct vision was presented by Winslow.[1] O'Dwyer's invention of his method of intubation in 1885 was an advance in treatment. Simpson[4] reported a case in 1908 of a patient who wore a hard rubber intubation tube almost continuously for over four years, and was successfully cured of a severe stricture. The core mold treatment of Jackson has been a great development, and with sufficient patience almost any stricture can be cured by this method.

The modern refinements in surgery: specifically, grafting of tissues, control of infection by antibiotics, and especially the development of plastics to be used for stents and molds, have increased the otolaryngologist's repertory to where many cases are cured by one or two stage operations. However, the results are still far from perfect in most cases, and there is still a great need for improved methods of treatment.

In treating an individual case, there are several procedures from which to choose, depending on various factors. Treatment by dilatation or by use of core molds requires frequent visits over many months. Although the results will be good in the end, most patients' circumstances are such that it is almost impossible for them to devote that much time.

Most patients are best treated by an external approach. If a tracheotomy is not already done, this should be the first step, and should be placed as low as possible. A general anesthesia may then be given and the thyrotomy done. The cricoid and trachea may also be opened if necessary.

The situation may be re-evaluated at this point and an appropriate procedure decided upon. If the obstruction is caused by webs between the vocal cords, these may be cut and a tantalum keel introduced between the vocal cords after the technic of McNaught. This keel is folded over on the outside of the thyroid cartilage to hold it in place. If, after the webs are cut and scar tissue excised, there still appears to be insufficient airway due to some distortion of the cartilages or fixation of the cords, we prefer to use a bone graft between the two alae of the thyroid cartilages and an acrylic stent as described by Woodward.[5] The body of the hyoid is easily exposed at the top of the thyrotomy incision and a section is taken from its center to be used as the graft. Small holes are drilled in the edges of the graft to facilitate suturing and the section of bone is then placed between the alae of the thyroid cartilage and sutured in place with chromic sutures. An acrylic reproduction of an O'Dwyer intubation tube must be inserted into the lumen of the larynx and secured by tantalum wire before the graft is sutured in place.

The acrylic stent can be made by any good dental laboratory if it is provided with a model. It is very important to have a lumen in the stent as this adds greatly to the comfort of the patient. Stents rarely cause any reaction or discomfort and may be left in place as long as necessary. Six months is an arbitrary time to plan for in most cases.

The hyoid bone graft may also be used to increase the lumen in the cricoid area in the same manner as above.

For very extensive strictures, especially when the trachea is involved, the skin grafting technic as described by Erich[6] is an appropriate solution. If this type of operation is necessary, the tracheotomy should be large and at least 1 to 2 cm. below the stricture. The stricture is exposed by a midline incision through the larynx and trachea and is excised. A split thickness skin graft is then obtained and sutured around a cylindrical piece of foam rub-

ber sponge with the raw surface outward. This is then inserted into the area of denuded epithelium and held in place by heavy silk sutures that go through the rubber mold and are tied externally across the skin incision. A heavy silk suture is also placed through the sponge rubber and brought down through the tracheostomy to be tied to the tracheotomy tube. After 10 days, the foam rubber mold is extracted through the tracheostomy. A model for an obturator is then constructed from an appropriate size rubber tube and dental compound. This obturator must have a lumen, fit into the grafted area, and have a lip that protrudes out through the tracheostomy and still leave room for a tracheotomy tube. After the model is constructed, the sponge rubber is reinserted through the tracheostomy into its original place until a dental laboratory can construct the obturator in acrylic. After this is obtained the rubber mold is again withdrawn and the acrylic obturator inserted into its place. It should be left in place for at least 6 months; usually this may be done without discomfort or need for special care. If the lumen is still adequate after the obturator is withdrawn, the tracheotomy may be closed. If the tube is left out for about a month, the stoma will close partially and then require only a simple procedure to complete the closure. Occasionally the stoma will be so large as to require multiple stages to close it.

Sometimes one finds the trachea completely separated from the larynx and then they have to be mobilized and anastomosed. An acrylic stent should be used to prevent stricture at the anastomosis.

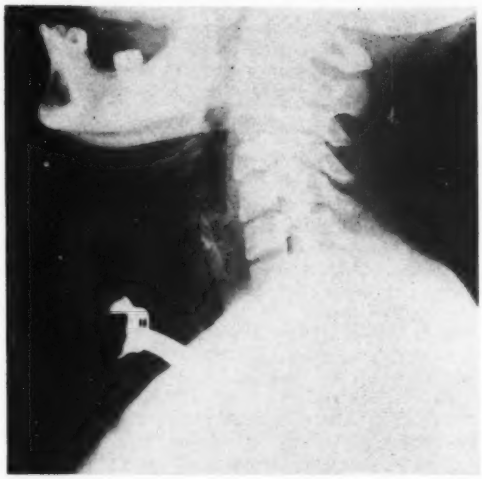
Case Reports

Case 1. A 23 year old man had worn a tracheotomy tube and had been unable to get any air through his larynx since an automobile accident 3 years previously. He was referred to the Ponce de Leon Infirmary where examination and x-ray studies showed a complete stricture at the cricoid and upper trachea and a bilateral paralysis of the vocal cords.

The tracheostomy was enlarged and the larynx opened. A complete stricture was found, as well as destruction of the anterior half of the cricoid and loss of at least one tracheal ring. The trachea was dissected free downward, brought up and anastomosed to the remainder of the cricoid. A section of the hyoid bone was grafted to bridge over the defect in the cricoid, and an acrylic stent was secured in place by a tantalum wire. He was able to talk for the first time in 3 years. After about 6 weeks he coughed up the acrylic mold. (See Fig. 1.)

The lumen shrank down as would be expected and on reopening the larynx about 2 months later he was found to have an epithelized lumen about 4 mm. in diameter. This was a big improvement over his original state, however. The Erich technic of skin grafting was then done after the scar tissue was excised. In an attempt to avoid the necessity of an arytenoidectomy later for the paralyzed vocal cords, a portion of the vocal cords was removed submucosally and the acrylic obturator was constructed long enough to fit between the cords. (See Fig. 2.)

FIG. 1



Roentgenogram showing an acrylic stent in the larynx. This patient had a section of hyoid bone grafted between the alae of the thyroid cartilages (Case 1).

FIG. 2

Acrylic obturator in place after skin grafting (Case 1).

After 6 months the obturator was removed. He had a good lumen and the vocal cords remained fixed in abduction. The large tracheostomy was closed in one stage with inversion of the skin about the edges and a sliding flap. A check-up one month later showed him still to have a good airway and he had resumed his job of bricklaying. This patient lives in Florida and we have not been able to get a follow-up report on him. I believe he would be back, however, if the airway were becoming inadequate. He was decannulated about 3 years ago. (See Fig. 3.)

Case 2. A 45 year old woman had several operations done for correction of a bilateral abductor paralysis of the vocal cords. She developed adhesions between her vocal cords and remained dependent upon her tracheotomy. A thyrotomy was done, the adhesions divided, and the Woodward technic of grafting a piece of hyoid bone between the alae of the thyroid cartilage and leaving an acrylic stent in the lumen was done. The stent was removed after 6 weeks and she was able to be decannulated a month later. Four years after operation, she is still able to do well without a tracheotomy. I believe she would have more lumen, however, if the stent had been left in place longer.

Case 3. A 35 year old woman had remained dependent upon a tracheotomy 3 months following an automobile accident. A thyrotomy was done and there was found to be a diminished anteroposterior diameter presumably due to fracture of the cartilages. The lumen was increased by using a bone graft between the alae of the thyroid cartilages and an acrylic stent was left in place. After 2 months the stent was removed endoscopically. She developed a cutaneous fistula and granulation tissue in the anterior commissure which promptly subsided after removal of a small sequestrum of bone through the laryngoscope. She was then decannulated and the tracheostomy allowed to close spontaneously. Three years after operation she does well except for some dyspnea on exertion.

FIG. 3

Roentgenogram after the obturator was removed and tracheostomy closed. This patient originally had a complete stricture (Case 1).

Case 4. A 45 year old woman developed a chronic stricture following removal of a tracheal tumor 10 years previously. On opening the trachea, a stricture of scar tissue about 1 cm. long and with a lumen of only about 3 mm. in diameter was found. The scar tissue was excised, and the Erich technic of skin grafting and construction of an acrylic obturator was done. The obturator was left in place for 6 months, then removed through the tracheostomy. Examination with a small mirror in the tracheostomy showed the lumen to be adequate and lined with epithelium. The tracheotomy tube was removed and after a few days the opening had contracted enough so a simple closure of the stoma could be done. Three years after removal of the obturator, she still has a good airway and no trouble with crusting.

Case 5. A 21 year old man had a severe injury to his neck in an automobile accident—a deep transverse laceration involving the larynx and pharynx was reportedly present. This was closed primarily and a tracheotomy was done. A subdural hematoma and multiple fractures prevented further attention. We saw him about 6 months after the injury, at which time the tracheotomy was closed up, but he was having a good deal of dsypnea on exertion and had a very poor voice.

Examination showed a markedly decreased glottic lumen with distortion and projection of a sharp ledge into the lumen. A tracheotomy was done and the larynx was opened. The projection visible on examination by mirror was found to be caused by a piece of cartilage. This was excised and a section of hyoid bone was removed and grafted between the alae of the thyroid cartilages after an acrylic stent was secured in place within the lumen. The acrylic stent was removed via the laryngoscope 6 months later and he was decannulated a month later. He is much improved but still cannot do heavy labor. However, he is able to do the job that he was trained for and is doing well.

Conclusion

A discussion of the causes and clinical characteristics of laryngeal strictures, some of the problems involved in treatment, a description of several methods of treatment, and case reports have been presented. Almost all cases can eventually be decannulated if the surgeon is persistent and patient.

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Roentgen Pelvimetry: A Precise Position Method*

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A. LOUIS DIPPEL, M.D., *Houston, Tex.*,
and COL. WOODROW L. PICKHARDT, MC,† *Fort Sam Houston, Tex.*

After reviewing the indications for x-ray pelvimetry and the deficiencies of current methods, the authors describe the advantages of their technic.

EVERY BOTTLE OF BLOOD is a bottle of dynamite, according to Unger,[1] of New York University, because some 2,500 Americans will die this year from the effects of blood transfusions. This creates a dilemma. No physician would suggest that we stop using blood transfusion because we all know that its advantages far outweigh its dangers. Instead, we must use blood only when actually necessary or beneficial, and then with the utmost discretion and care.

A similar dilemma has arisen regarding x-ray pelvimetry because of the hue and cry about the dangers of diagnostic x-ray examinations. One of the results may be an injudicious reduction in the use of x-ray pelvimetry with an increase in unnecessary cesarean sections and an increase in fetal and maternal damage from traumatic delivery in unrecognized disproportion. We are convinced that x-ray pelvimetry should be used just as transfusions, without fear, when necessary or helpful, and with care and discretion.

Some years ago DeLee[2] said: "Since the x-ray cannot measure the strength of the pains or the moldability of the head, or the expandability of the pelvic joints, or the mutation of the sacrum, or the rigidity of the soft parts, or the metabolism of the parturient, and so forth, you can see what a small role it plays in the routine conduct of the delivery, but I would not be without it."

Eastman[3] said that x-ray pelvimetry is essential to the intelligent management of cases of pelvic contraction, but that its introduction the country over has been more harmful than helpful. He attributed this to faulty interpretation of pelvimetry films and reliance on specific pelvic measurements, to the subordination, or even utter neglect, of the over-all clinical picture. This subordination usually is due to the obstetrician's failure to acquaint himself with x-ray pelvimetry and his acceptance of the interpretation of the roentgenologist who is not trained either to advise in the management of labor or to predict the final outcome.

Indications

There is much more to x-ray pelvimetry than the mere determination of certain measurements. It affords the only reasonably accurate estimate of the fetal size, not in pounds, but in relation to the maternal pelvis, malposition, engagement, molding, descent and the architectural details which have more effect upon labor than do the "diameters" except when the "diameters" are very long or very short. Steel and Javert[4] found that the clinical measurement of the inlet revealed only one half of the contracted pelves. Dippel[5] proved that the obstetric conjugate determined exactly by x-ray disagrees widely with the obstetric conjugate estimated clinically from the diagonal conjugate, especially when the latter measured 11.5 cm. or less—that is, within the contracted range. He concluded that the patient with a diagonal conjugate of 11.5 cm. or less should be given the benefit of x-ray pelvimetry because, in this range, precise information is invaluable.

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It is obvious, then, that x-ray pelvimetry is essential for adequate evaluation and proper management of certain obstetric cases. It should be done at term, or ideally, upon admittance to the labor room, on patients with:

(1) small pelvis by clinical measurement,

(2) suspected fetopelvic disproportion,

(3) a history of difficult delivery, unexplained stillbirth, or pelvic injury,

(4) on primigravidas at term with the presenting part unengaged,

(5) on primigravidas whose height is 5 feet or less, and

(6) in all cases of expected or unexpected dystocia.

A lateral "progress" film should be taken during labor (and repeated as necessary) on any patient whose progress is unusually slow or delayed.

Unfortunately, few obstetricians utilize the information obtainable from x-ray studies in suspected disproportion. From a survey of the use of x-ray pelvimetry by southeastern obstetricians, Jones[6] found that most obstetricians are so disinterested that they do not learn what is a good technic nor what to expect from it. They prefer the radiologist to give them measurements, which are of little value unless combined with the obstetrician's appraisal of the films.

When intelligent interpretation of pelvimetry films is made by the obstetricians, the cesarean section rate is low. At the New York Hospital routine x-ray pelvimetry in certain groups lowered the gross fetal mortality and the rate of cesarean section done for disproportion.

Stander[7] believed that the use of x-ray pelvimetry would lead to an over-all reduction in the cesarean section rate and it is being used more frequently for correct indications and less frequently for the incorrect indications.

The judicious use of x-ray pelvimetry at Brooke Army Hospital and our insistence that all the obstetric staff become expert in the interpretation of the films, together with our study of vaginal delivery following previous cesarean section[8] probably account largely for the cesarean section rate of 0.94% for the last 24,000 deliveries.

Technic

In a critical review, Hodges and Dippel[9] considered most of the methods of x-ray pelvimetry sufficiently accurate for clinical purposes, but not exact. They criticized the isometric grid methods because correct placement of the grid is necessary and in many cases may be impossible. In most of the position technics, accuracy is dependent upon the proper positioning of the patient and the proper positioning of the markers.

These factors presented a real problem at Brooke Army Hospital. X-ray pelvimetry can be obtained at any time, day or night. The films are taken by any of several technicians and are interpreted by the obstetrician. The films are then kept on the labor room floor for ready reference until the patient has been delivered. By the isometric method formerly used, 30 to 40 minutes were required for each case.

Largely because poor positioning of the patients resulted in films which did not portray a true lateral or frontal shadow of the pelvis, there was a 40 to 50% "retake," with an increase in cost and radiation exposure of the patient and fetus.

We planned to devise a technic which would:

(1) be precise,

(2) have *small* and *measurable* limits of error,

(3) be time saving,

(4) require only simple and exact positioning of the patient and markers so the x-ray technicians could quickly and easily become proficient in its application, and

(5) not be costly.

It was known that obtaining accurate anteroposterior diameters from a *true* lateral film of a symmetrical pelvis is easy. If a known marker is placed between the thighs close to the symphysis pubis, it lies in the sagittal plane of the pelvis and is distorted similarly as any anteroposterior diameter of the pelvis in the same plane.

There are several pitfalls in taking this film. If the patient is lying on her side or is standing, it is difficult to obtain a true lateral film, and it is the exception rather than the rule when the shadows of the

acetabula are directly superimposed. But, if we have the patient sit on a table, with one hip close to the lateral cassette, rotation of the pelvis is minimal and a true lateral film is the rule (Fig. 1).

In other position methods, the measurement of the transverse diameter of the inlet and the interspinous and intertuberous distances from the frontal film presents a more difficult problem. Attempting to place markers at the level of these diameters to achieve equal distortion is pure guesswork. The placement depends entirely upon palpation and *can be* only approximately accurate even when done by a highly trained technician.

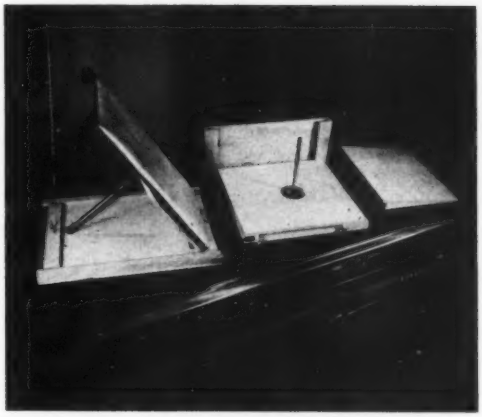
In computing these diameters, we must consider distortion. When the target film distance and the object film distance are known, the amount of distortion of an object on an x-ray film, with the object parallel to the film, can be computed accurately with the formula:

$$O = S \frac{D\text{-}d}{D}$$

O is the true size of the object; S, the size of the shadow on the film; D, the target film distance, and d, the object film distance. The principal criticism of many position technics is that none provides a method to determine accurately the object film distance.

An inexpensive table was constructed to support two cassettes, one perpendicular to the other. It was constructed in three parts for easy storage and light enough for a female technician to assemble easily (Fig. 2).

FIG. 2

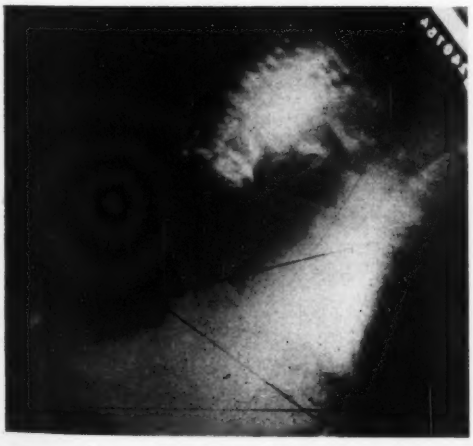


Table—unassembled.

The patient sits over a 10 by 12 inch cassette for taking the frontal film with the known marker between the thighs near the symphysis pubis and *perpendicular* to the cassette upon which she sits (Fig. 3). The distance from the zero notch of the marker to the frontal film is known and constant. The lateral film is taken on a 14 by 17 inch cassette, perpendicular to the frontal film and *parallel* to the marker (Fig. 4).

A 6 inch straight cone is used for both films to improve detail and to reduce the exposure of the patient to x-rays. The central

FIG. 1

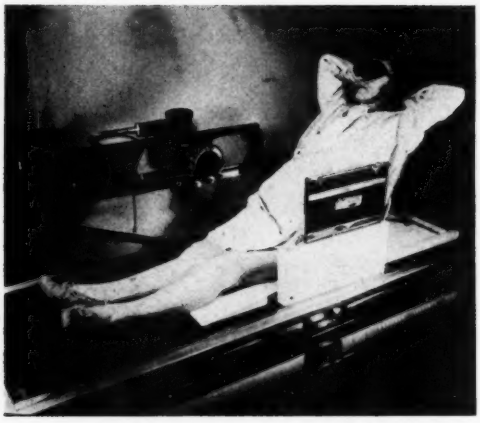


A good lateral film.

FIG. 3

Position for exposure of frontal film.

FIG. 4



Position for exposure of lateral film.

ray is aimed through the acetabula for the lateral view. For the frontal exposure, it is aimed over the pelvis, slightly posterior to the anterior superior spines. The patient does not move between exposures of the frontal and lateral films. *This is important.*

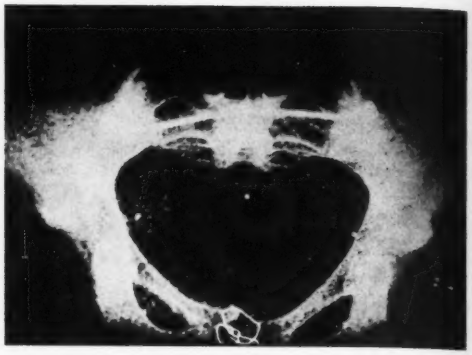
The positioning of the patient and the placement of the marker are simple and easy for the technicians. They must use a constant target film distance (we use 42½ inches or 106.68 cm.) and be certain that the central rays are perpendicular to the film. An average of 8 to 12 minutes is required to take a frontal, lateral, and soft tissue lateral for visualization of the placenta when the last is desired. Few "retakes" are required. This is in sharp contrast to the isometric methods formerly used.

Determining the anteroposterior diameters of the pelvis from the lateral film is easily accomplished with calipers, utilizing the distorted shadow of the marker as the centimeter scale (Fig. 1).

Measurement of the transverse of the inlet, the interspinous and the intertuberous diameters taken from the frontal film can be precise but requires some effort. Since the base of the marker is a known distance from the frontal film, the object film distance of the diameter to be measured from the frontal film can be found by referring to the lateral film.

For example, the end points of the spines are located and the distance between them

FIG. 5



Roentgenogram of dried pelvis in rice.

measured. On this film it was 11.9 cm. (Fig. 5).

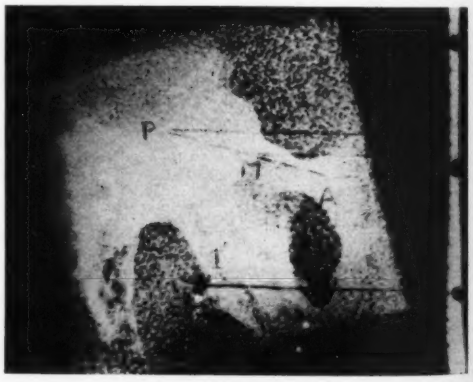
From the lateral film we see that the projection of the interspinous plane transects the shadow of the marker at 4.9 cm. (Fig. 6). To this is added 2.2 cm., the known distance from the base of zero notch on the marker to the film. Therefore, 7.1 cm. is the object film distance for the interspinous diameter.

From the equation: $O = S \frac{D-d}{D}$

We learn that: O = 11.09 cm., the actual measurements of the interspinous distance.

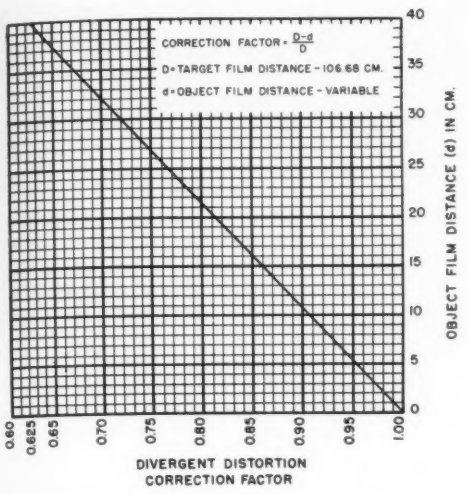
If one prefers a mathematical short cut, a graph may be made (Fig. 7) to find the distortion correction factor for any object film distance when the target film distance is

FIG. 6



Roentgenogram of dried pelvis in rice.

FIG. 7



Divergent distortion correction factor graph.

known and *remains constant*. Since the object film distance of the interspinous diameter has been found to be 7.1 cm., we find from the divergent distortion graph that the correction factor is 0.932. The measurement of the interspinous diameter on the frontal film, 11.9 cm., multiplied by the distortion factor 0.932, gives us 11.09 cm., the actual interspinous distance. Other transverse diameters can be computed in the same way.

We want to emphasize that the roentgenologist can report only what he actually sees and measures: an accurate, *quantitative* evaluation of the pelvis. On the other hand, the obstetrician, by combining this accurate quantitative information with his clinical knowledge of the patient, can determine many qualitative factors which, more often than not, are of greater value than measurements alone. By careful study of good films, he easily can assess the relative sizes of the fetus and pelvis, engagement, descent, and molding. Other qualitative factors, such as the type of pelvis, the depth and width of the sacrosciatic notch, the width, curvature, inclination and thickness of the sacrum, the internal angle of the fore pelvis, the inclination of the symphysis, the splay of the side wall, and the presence or absence of "funnelling," enable the obstetrician to anticipate the mechanism of labor and to plan intelligently his conduct of the labor and execution of the delivery. The coccyx is unimportant and is considered a part of the soft tissues. This information cannot be expected from the roentgenologist.

The qualitative information obtained from x-ray studies of the pelvis has changed the concept of indications for operative obstetric technics.

It is essential to know in what diameter the after-coming head of a breech can best enter the pelvis. It is important to know whether it is better to deliver an occiput posterior as such or to rotate it, and at what level the rotation should be accomplished.

To prove the accuracy of this technic, measurements from the x-ray films were compared with those on dried pelves (Fig. 5). Lead shots were fixed to the commonly used "end points" with cellulose tape. The pelvis was embedded in dried rice in a cardboard carton to simulate the absent soft tissue and radiographic pelvimetry was done by our method on four occasions. Three independent observers were given the pelvis and asked to make ten measurements of the distance between the shot marked "end points." An average of ten readings for each measurement was taken. Later, three other independent observers measured these diameters on the x-ray films and averaged their findings. By comparison of the direct measurement on the dry pelvis and those on the films we found an error of no more than 0.4 to 0.6 mm. for any diameter. This is an error of well under 1%, which we consider acceptable. It was the opinion of the observers who did the measurements that the radiographic measurement is more accurate than the direct measurement.

Summary

(1) A technic of x-ray pelvimetry developed at Brooke Army Hospital has been described. It offers precision, simplicity, low cost, it is not time-consuming, and exposes the pregnant woman and her fetus to a relatively small amount of irradiation.

(2) Quantitative interpretations have been discussed.

(3) The value of the qualitative factors which the obstetrician should learn from the films has been stressed.

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Neurologic Manifestations of Sarcoidosis

WILLIAM H. GOODSON, JR., M.D.,† *Nashville, Tenn.*

In some one to five per cent of cases of sarcoidosis there is clinical evidence of involvement of the nervous system, more often of the cranial and spinal nerves. There are instances of meningeal invasion and tumor formation. Primary disease of the muscles may be encountered.

THE FIRST RECORDED CASE OF SARCOIDOSIS was probably the one described by Hutchinson in 1869, in which the skin lesions were delineated and distinguished from those of lupus vulgaris. In 1889, Besnier reported a patient with similar cutaneous lesions and named the malady "lupus pernio." A case thought to be of the same nature as Hutchinson's was described by Boeck in 1899, and given the name "multiple benign sarcoid of skin." These clinical disorders were given a common pathologic denominator in 1914 by Schaumann, who referred to the condition as "lymphogranulomatosis benigna." Pulmonary lesions were described in 1915 by Kuznitsky and Buttorf, and the radiologic aspects of osseous involvement were presented by Jüngling in 1928. It was not until 1936 that the "febris uveo-parotidea subchronica," given a place in clinical medicine by Heerfordt in 1909, became established as a part of the disease spectrum. Today this patchwork of observation has been assembled into the well-known entity, sarcoidosis, a disease of baffling etiology and protean manifestations.[1]

Historical Aspects

Most of the early reports of involvement of the nervous system are found among recorded cases of the uveoparotid syndrome. In 1909 Heerfordt described craniospinal nerve deficits and diebetes insipidus in association with uveoparotid fever. Before him, Daireaux (1899) had reported a case of "mumps neuritis," probably an instance of sarcoid. Similarly, in 1923, MacBride[2] in a paper entitled "Uveoparotitic Paralysis" described a case of peripheral neuritis, followed by uveoparotitis and palsies of the fifth and tenth cranial nerves. He considered such etiologic possibilities as mumps, Hodgkin's disease or some toxic agent. Other case reports of polyneuritis and uveoparotitis were presented as possible instances of "atypical," noncaseating tuberculosis. It soon became recognized that there were abnormalities of the nervous system in about 50% of the cases of Heerfordt's disease, ranging from the more frequent cranial nerve palsies to peripheral neuritis, diabetes insipidus, hypersomnia, spinal cord involvement, and cerebellar deficits. Pleocytosis and increased protein content of the cerebrospinal fluid had also been noted. Waldenström[3] (1937) reported a case of uveoparotitis with signs of brain stem, cortical, cerebellar, meningeal, and peripheral nerve involvement.

To a lesser extent reports of cases of lymphogranulomatosis benigna (Schaumann's disease) with neurologic manifestations were accumulating in the medical literature. Roos[4] reviewed this subject in 1940 and found 12 such cases, 8 with cerebral symptoms, 2 with peripheral neuropathy, 1 with optic neuritis, and 1 with facial paralysis. Pleocytosis and elevated cerebrospinal fluid protein were also noted. At autopsy there was involvement of the central nervous system in 3 of them.

More recent reviews have indicated that sarcoidosis may involve the nervous system in 1 to 5% of cases. In reviewing 160 cases of sarcoidosis, Longcope[1] found 2 such instances, one with diabetes insipidus and one with neuropathy. Other investigators reported an

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incidence of involvement of the nervous system 7 in 150 and 2 in 300 cases.[5] Ricker and Clark[6] found 2% in 300 cases. It is generally agreed that lesions of the nervous system are more frequent in the uveoparotid syndrome than in the other "types," although they may be present with no evidence of sarcoid elsewhere.[7,8] In a survey of 118 cases with neurologic manifestations, Colover[9] found the frequency of associated features to be that shown in table 1.

Neuromuscular Lesions

Neuromuscular lesions may involve, (1) craniospinal nerves, (2) central nervous system (brain, spinal cord, meninges), or (3) muscles.

(1) Craniospinal Nerves.

Cranial nerve lesions are most frequently encountered, and of these the seventh nerve is most often involved. Colover found facial paralysis in 58, or 50%, of 118 cases. This was unilateral in 65% of patients, and in the remaining 35% bilateral paralysis ensued after an interval ranging from days to months. Facial paralysis may follow a parotitis by a few days to 6 months, precede it, or occur independently of parotid involvement. It is usually of sudden onset and self-limited, although sequelae such as contractures or spasms sometimes occur. Matthews[10] reported such residua in 4 of 6 instances of facial paralysis due to sarcoid. The pathogenesis of this facial palsy has been debated. Its frequent association with parotitis suggests such factors as inflammation or pressure due to an edematous parotid gland. When no parotid swelling is obvious, it has been proposed that hidden swelling in the region of the stylomastoid foramen might be present. The regression of facial paralysis at a time when parotid enlargement may be progressing militates against this explanation. Furthermore, the finding of an associated ageusia, which may occur in 18% of cases with facial palsy,[9] is not consistent with this explanation, since this feature places the lesion farther proximally than the stylomastoid region.

TABLE 1

FREQUENCY OF ASSOCIATED CLINICAL FEATURES IN 118 CASES OF SARCOIDOSIS WITH NEUROLOGIC INVOLVEMENT (FROM COLOVER[9])

	Number of Cases	*% of Cases*
Parotitis	73	62
Iridocyclitis	67	56
Pyrexia	44	37
Cutaneous lesions	33	28

All of the other cranial nerves have been affected. Colover found them to be involved in the following order of decreasing frequency: optic, glossopharyngeal, vagus, acoustic, oculomotor, trigeminal, hypoglossal, olfactory, abducens, accessory, trochlear. The most striking lesion of the optic nerve was found in a case in which there was a sarcoid tumor mass in the optic nerve causing 20 diopters of papilledema. Matthews' case 1 was characterized by paralysis of all cranial nerves except III, IV, and VI, within a six week period. These multiple cranial nerve palsies, like the isolated facial nerve involvement, tend to resolve spontaneously.

Peripheral neuropathy is not as common as cranial neuropathy. Neuritic pain is a frequent symptom, and may be symmetrical (resembling acute infectious polyneuritis) or asymmetrical. Other sensory and motor phenomena common to neuropathy also occur. Recovery ensues usually within weeks or months but may not be complete, and relapses also occur. The gross lesions are irregular or spindle-shaped swellings of the affected nerves, with the typical granulomatous sarcoid histopathologic pattern.[9]

(2) Central Nervous System.

Sarcoid may involve the central nervous system (brain, spinal cord, and meninges) in a small percentage of cases. In the Ricker and Clark study, of 195 cases of known organ involvement (excluding lymph nodes), 3 or 1.5% had lesions of the brain and meninges. In 1951, Pennell[11] was able to list 51 reported cases with involvement of the central nervous system out of 1,500 sarcoid cases in the literature and added 3 more of his own. In 20 cases of sarcoidosis proved at autopsy Aszkanazy[7] found 3 with disease of the central nervous system. The clinical picture presented in these cases depended upon the location of the lesions and the degree of obstruction to circulation of the cerebrospinal fluid.

The most frequent site of involvement of the central nervous system is the base of the

brain, in the form of a granulomatous or adhesive arachnoiditis, or an invasion of the floor of the third ventricle.[7,12,13] Grossly, the lesions may be too small to be seen, or they may be mistaken for tumor. Microscopically, the picture is identical with that of sarcoid anywhere in the body. (Aszkanazy, however, reported no inclusion bodies seen in his series.) There is a localized destruction of tissue with secondary degeneration of any tracts involved. Direct invasion of the brain from the subarachnoid space is not uncommon (as opposed to tuberculosis). Common sequelae of basal involvement are optic atrophy, compression of other cranial nerves, obstructive hydrocephalus, and pituitary or hypothalamic disturbances. Colover collected 20 cases with polyuria and polydipsia, 6 cases with hypersomnia and lethargy, and many others with obesity, hypogonadism, amenorrhea, bitemporal hemianopia, or enlargement of the sella turcica, all signs referable to the hypothalamic-pituitary region. Pennell found diabetes insipidus in 18 of 51 cases, and 2 of these also presented the adiposogenital syndrome. In 3 other cases only the latter was seen. Three cases have been reported in which an adhesive arachnoiditis of the posterior fossa blocked the foramina of Luschka and Magendie, simulating a posterior fossa tumor.[9,11] Another case, simulating a space-taking lesion, was seen by Meyer, in which at autopsy a diffuse granulomatous angiitis and periangiitis of the leptomeninges was seen.

There have been 7 cases reported in which clinically and operatively the lesion was a circumscribed tumor in the central nervous system.[5,7,8,14] These have been found in the floor of the third ventricle, occipital, frontal and temporal lobes, inferior horn of the lateral ventricle, subfrontal area, and the floor of the middle fossa. In Goodman's case[8] the tumor arose from the subfrontal area and extended to the longitudinal sinus and falx, involving "en plaque" the dura of the convexity of the frontal lobe. In two cases the tumor mass was thought at operation to be a meningioma. Saltzman[15] reported radiologic findings in 5 cases of tumor-like Boeck's sarcoid. The ventriculograms were similar to those of any neoplastic process. Angiography in two cases revealed poorly vascularized lesions. In two cases that were followed without operation, local atrophy developed following regression of the lesions.

The spinal cord was involved in 10 reported cases.[16,17] In one a basal arachnoiditis extended into the spinal region through the foramen magnum. In another there was involvement of the sacral cord which regressed under observation. Aszkanazy found granulomatous nodules in the thoracic cord, spinal meninges, and nerve roots. In a patient with a sensory level at T-12 L-1 dermatome, Moldover[16] reported pronounced improvement after ACTH therapy. All but two of these 10 cases developed permanent neurologic damage.

When the clinical picture is that of a meningitis, it is usually of a chronic nature as described above. Naumann,[18] however, observed a 3 month old infant (the earliest age at which central nervous system sarcoid has been reported) with a fatal course of six weeks duration, characterized by convulsions, nuchal rigidity, pyrexia, anemia and pleocytosis. At autopsy sarcoid lesions were seen subdurally in the falx, tentorium, and under the sutures.

(3) Muscles.

Primary disease of the muscle due to sarcoid has been described in 53 patients, 25 showing clinical evidence of the disorder.[19] In 10 of these patients the pattern of involvement resembled muscular dystrophy with more implication of proximal than distal muscles. This involvement was symmetrical in all but 2 cases, and often resulted in fibrous contractures and muscle pains. In 2 patients there was an increase in muscle volume. Dysphagia was a feature of one case. Muscle biopsy at random in patients with sarcoidosis, but without muscle symptoms, has resulted in from 60 to 75% positive results, indicating a higher incidence of muscle involvement than is generally recognized.

Cerebrospinal Fluid

Alterations in the cerebrospinal fluid are found frequently in cases of sarcoid involving the nervous system except when the pituitary alone is involved. In the latter instance abnormalities of the cerebrospinal fluid are encountered infrequently. The commonest

derangement is an elevated protein, usually within the range of 100 to 400 mg. per 100 ml., with the highest value reported being 860 mg. per 100 ml. (Cares[13]). One review reported the protein elevated in 80% of cases, with a median value of 275 mg. per 100 ml. Another common finding is a moderate pleocytosis of 10 to 60 cells per cu. mm., although counts up to 162 cells per cu. mm. have been reported.[9] Lymphocytes usually predominate. In the few cases in which the sugar content has been reported, it was abnormally low, in the range of 10 to 40 mg. per 100 ml. in about half of the studies.[8,11] The pressure of the fluid may be moderately elevated. Abnormal first-zone or mid-zone colloidal gold curves have also been observed.[8]

Treatment and Prognosis

The treatment of sarcoid of the nervous system, as with sarcoidosis in general, is not satisfactory. Reports of successful results with steroids are occasionally seen,[16,19,20] but on the whole this therapy has not been effective.

The prognosis, in general, appears to be poorer if there is involvement of the nervous system, especially the central nervous system. In one series of 140 cases of sarcoidosis it was found that 48% died as a result of sarcoid, and in 25% of those fatalities there was nervous system involvement.[5]

Clinical Material

All of the cases of proved sarcoidosis from the pathology files of the Vanderbilt University Hospital and the Thayer Veterans Administration Hospital were reviewed with particular reference to neurologic manifestations. A total of 63 cases were studied, of which 60 were proved by biopsy and 3 proved by autopsy. There were 38 males and 25 females, with an average age of 31 and an age range of 13 to 63 years. Thirty-six were Negro and 27 white.

Case Reports

There were 4 cases with findings pointing to nervous system involvement, and one case with primary muscle involvement.

Case 1. P. A., a 32 year old colored woman, was seen on Jan. 6, 1956, in the Vanderbilt University Outpatient Department with a one week history of parotitis and facial weakness on the left, weight loss, dysphagia and dysarthria. She was found to have left seventh, ninth, and tenth cranial nerve paralyses. Three weeks later the only remaining deficit was a partial facial paralysis.

She presented with sarcoid skin lesions 2 months later and subsequently developed lymphadenopathy, cough, hepatosplenomegaly, leukopenia with 10% eosinophils, and a reversal of the serum albumin-globulin ratio. Roentgenograms of the chest revealed lesions compatible with sarcoid. Skin and lymph node biopsies were diagnostic of the disease. Cultures of sputum and gastric washings were negative for tubercle bacilli. Old tuberculin and histoplasmin skin tests were negative. She had a positive serum Wassermann test. Upon examination of the spinal fluid the protein was 20 mg. per 100 ml., there were 3 mononuclear cells per cu. mm., and the Wassermann was positive in all dilutions. In July 1956, there remained a slight residual of the facial paralysis. By July 1958 there was a complete remission of the clinical and radiologic abnormalities, without specific therapy.

Case 2. J. E., a 22 year old white man, presented himself at the Thayer Veterans Administration Hospital in 1948 with a 2 week history of headache and aching pain in the left side of the face, with paresthesias involving all three divisions of the trigeminal field on that side. The positive findings on examination included generalized lymphadenopathy, hypesthesia in the distribution of the left fifth nerve without a depressed corneal reflex, and a mild left facial weakness. Roentgenograms of the chest and hands were typical of sarcoid, and a lymph node biopsy was diagnostic. The neurologic symptoms disappeared within 7 weeks.

Case 3. F. M., a white woman, had a rash, lymphadenopathy, and fever as well as a positive node biopsy for sarcoid in 1952, at the age of 35. Following a remission she developed, in July 1955, frontal and occipital headache, dizziness, nausea and vomiting, and diminished visual acuity in the right eye. She was treated with steroids followed by a brief remission until September 1955, when steroids were again administered due to a recurrence of these symptoms. She was admitted to Vanderbilt University Hospital for evaluation in October. On physical examination she was found to have a skin rash, generalized lymphadenopathy and hepatomegaly. The neurologic examination at this time was within normal limits. Two cerebrospinal fluid examinations revealed 40 and 104 leukocytes per cu. mm. (66% mononuclear), with a protein content of 105 and 112 mg. per 100 ml. and a sugar content of 37 and 30 mg. per 100 ml. The cultures were sterile and guinea pig inoculations were negative. The spinal fluid Wassermann test was nonreactive and the electroencephalogram was within normal limits. Three gastric washings were negative for acid fast bacilli. Histoplasmin, old tuberculin, and coccidioidin skin tests were negative. She left the hospital improved without specific therapy.

Case 4. K. F., a 13 year old boy, was admitted to Vanderbilt University Hospital in March 1958 because of a gait disturbance which began 5 weeks prior to admission. His parents had observed that he shuffled his feet as he walked, and he had complained

of pain in the calves. He tired easily and had lost 10 pounds in the 5 months before admission. The gait disturbance began to show improvement before admission to the hospital. No neurologic abnormality was found on admission, and it was thought that he might have had transient bilateral involvement of the peroneal nerve. A chest film revealed hilar masses. Skin tests (old tuberculin and histoplasmin) were negative. The spinal fluid was normal except for a mild pleocytosis (14 mononuclear cells per cu. mm.). An exploratory thoracotomy was performed, and the hilar masses were found to be enlarged lymph nodes with the histologic pattern of sarcoid (Fig. 1).

Case 5. M. D., a 21 year old colored man, was seen at another Veterans Administration Hospital in 1945 with generalized muscular aching, fever, conjunctivitis and parotid swelling. He was found to have multiple nodules in his muscles and tendons without atrophy, and roentgenographic evidence of a lung infiltrate consistent with sarcoid. Biopsies of a lymph node and a muscle nodule revealed typical sarcoid lesions. No treatment was instituted. Two years later he was admitted to the Thayer Veterans Administration Hospital with visual impairment found to be related to a healed iritis. At this time, however, there were no signs of active sarcoidosis.

Comment. The typical cranial nerve involvement is exemplified by cases 1 and 2. Case 1 was a diagnostic problem for 3 months because the full-blown clinical picture of sarcoidosis was preceded by the neurologic symptoms. The cranial nerve paralyses can be assumed to be unrelated to the syphilis on the basis of an inactive spinal fluid formula and the rapid recovery.

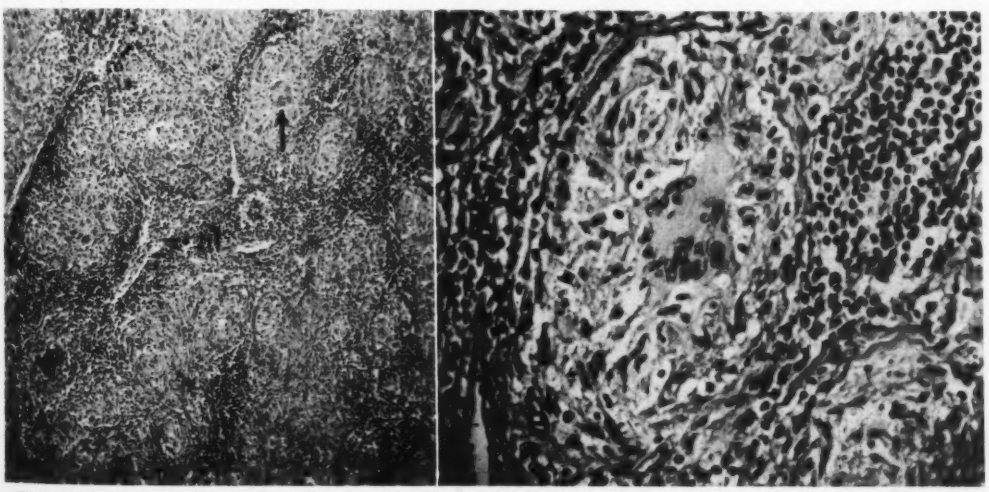
The third case presented meningitis which was suspected of being tuberculous in origin. However, all laboratory procedures failed to demonstrate evidence of tuberculosis. In the light of a biopsy diagnosis of sarcoidosis and subsequent improvement without therapy, it is suggested that she had a granulomatous meningitis in association with sarcoid.

In the fourth case it cannot be stated categorically whether the gait disturbance and pain were related to sarcoidosis of the nervous system, since the patient presented no abnormal neurologic findings on admission. However, a symmetrical peripheral neuropathy of the lower extremities, resolving within a period of five weeks, is consistent with a diagnosis of sarcoid neuritis.

Case 5 is presented not as a strictly neurologic problem, but to emphasize the fact that sarcoid can present as diffuse muscle disease. No muscle atrophy was present in this case, and it did not pose a problem in differential diagnosis of the various myopathies.

Accepting these 4 cases as being representative of the neurologic complications of sarcoidosis, an incidence of 6.3% is obtained for this small series, slightly higher than that usually reported in the literature. The distribution of the involvement is in accord with previous reports on this subject, with 3 instances of cranial or peripheral neuritis, and one case of meningitis.

FIG. 1



Photomicrographs from the hilar nodes in case 4. A (50 X) shows the replacement of normal architecture by epithelioid proliferation. The giant cell indicated by the arrow is shown in B (200 X).

Summary

1. Sarcoidosis involves the nervous system in 1 to 5% of cases. The most common manifestation is craniospinal neuropathy, but meningitis and tumor-like involvement of the central nervous system may occur, as well as primary muscle disease.

2. Five cases of sarcoidosis are reported, in 4 of which there was neurologic involvement, while one presented as a diffuse muscle disease.

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Patterns in the Distortion of Scientific Method*

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The author reviews approaches used in the investigation of schizophrenia in the past. He warns of the dangers of distortion of apparent scientific methods by preconceived thoughts and ideas. This is an interesting historical review.

INVALIDATED INVESTIGATIONS into the cause of schizophrenia are seldom regarded once they have been rejected. However, they should offer excellent sources of data to determine what patterns of distortion of scientific methods occurred. The identification of such patterns might prevent future failures.

The most readily available of such studies are those investigations which resulted in the concept that focal infection was the sole etiologic agent in schizophrenia. These studies received their greatest prominence during the period of about 1918-1924. I am well aware that a number of workers subscribe to a theory of bacterial etiology of schizophrenia at the present time. Yet, since about 1924, the profession has obviously rejected the validity of these investigations. Regardless of the etiology or etiologies of schizophrenia which may ultimately be identified, it is correct to say that the profession has considered the scientific method in these earlier investigations to have been invalid.

These investigations should be considered under three headings: first, the hypotheses and the extent to which they were accepted by, and influenced, American psychiatry, second, under what might be called the social scientific field, and third, the scientific field, *per se*.

The hypothesis in brief was as follows—that foci of chronic infection elaborated a toxic product or products, which were responsible for the symptoms of psychosis. Schizophrenia and manic-depressive illness appear to have been included by all who supported this theory. One author states "We will confine our statistics to the so-called 'functional group' in which we include manic-depressive insanity, dementia praecox, paranoid condition and the psychoneuroses."[1] Another included epilepsy as being similarly caused.[2] It followed therefore that these illnesses could be cured by surgical removal of the foci of infection, or if this were not feasible, through the use of autogenous vaccines. The exception was made that had the process gone on more than 2 to 3 years irreversible changes might have occurred in the brain. Foci were considered to be most frequently in the teeth, tonsils, sinuses, appendix, cervix, gallbladder, colon and terminal ileum, or the seminal vesicles. Failure of a patient to respond to treatment (in early cases) was accepted as evidence that undiscovered foci were still in existence and increasingly radical surgical operations were performed.

Some idea of the numbers of patients involved, and the drastic nature of the treatment procedures used can be seen from the two following excerpts. In 1922, one author, in an article entitled "The Etiology and Treatment of the So-Called Functional Psychoses" states, "the successful treatment of 1400 cases during the last four years with only 42 return cases now in the hospital must be accepted as evidence that our work has been efficient . . . the increase of 43 per cent (discharged) is due entirely to the method of detoxication employed."[1] Elsewhere he states that of these 1,400 cases only 62 had resections of the colon. Later, "while we had done many more operations on the colon it has been on the chronic cases with little or no results. Our statistics would be approximately as follows: In 250 operations, 62 have recovered, or 25

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per cent; the death rate has been 30 per cent; improved cases, 15 per cent; and 30 per cent unimproved and where there is no question as to the outcome of the psychoses, being terminal dementia, we feel justified in performing colectomy, with the possibility of recovery."

Another author refers to epilepsy as "chronic convulsive toxaemia" and reports a large experience "embracing in excess of a thousand cases, in all the diagnosis of abdominal conditions was confirmed by X-ray plates, aggregating several thousand, and several hundred by surgical exploration of the abdomen."[2] He refers also to similar confirmatory observations, being reportedly made at various hospitals. The English literature reveals similar interests and investigations at this time.[3]

There is ample evidence that the issue of the validity of these claims was of vital concern to the profession. Popular belief in the validity of these procedures placed the majority of psychiatrists under great pressure from relatives.

The degree of support which the theory of focal infection gained can, in part, be understood by considering the status of psychiatry and its relation to the scientific community at this time.

In 1894, Weir Mitchell had indicted psychiatry for, among other things, its isolation from medicine in general and for its lack of scientific method in its approach to mental illness.

By 1922 great advances had been made in medicine and surgery and none was more spectacular than in bacteriology. Science at this time appeared to consist of certain basic postulates and methodologies, which, if followed, could not but fail to lead to discoveries. One of the first requirements in method, that of classification, had been satisfied by Kraepelin, despite his procrustean methods. Second, the principle of unicasuality appeared to have been firmly established.

In patients suffering from general paresis a positive spinal fluid Wassermann had been demonstrated by 1906, and by 1913 Moore and Noguchi demonstrated *Treponema pallidum* in the cortex. In 1917, Wagner-Jauregg inoculated his first six patients with tertain malaria. Psychiatry had its first specific treatment, which had been achieved by following the accepted scientific methodology.

In general medicine a wide variety of illnesses were being attributed to focal infection, and the interest in this subject was intense. The Quarterly Cumulative Index, under the heading "Infection, focal," lists an average of 50 articles a year from 1920 to 1924 inclusive.

When these pressures are considered it seems almost inevitable that focal infection would be blamed as the etiologic agent in schizophrenia. Also this was a theory with a strong appeal for many psychiatrists.

Populations of mental hospitals were rapidly increasing at this time and it was becoming increasingly difficult to maintain even minimal standards of care. Many superintendents supported these theories with the avowed intention of using them in the hopes of obtaining adequate staffs and medical equipment from state legislatures.

Whatever attempts to promote their convictions the investigators may have made, the more important factor seems to be that they were driven by strong unconscious compulsions and, in addition, that these compulsions were shared by many psychiatrists. Only the sharing of these compulsions by other members of the profession—and to a greater degree by the lay public—could account for the rapid promotion of these concepts without an adequate investigation of their validity. The marked rapidity with which they were later dropped when conflicting data was presented certainly suggests that feelings of guilt and anger were being expressed.

Sources of error within the investigations can now be considered. Many of the methodological errors to be found were inherent in the design of the work. The design proceeded from the hypothesis that schizophrenia was a dementia. The philosophical position was stated "that there can be no disorder in function without a disorder in structure." In the case of one of the investigators this was further bolstered by his conviction that he had described hitherto unreported pathologic changes in the brains of schizophrenics.[4] Thus, the initial hypothesis was not merely that an underlying organic cause was more probable, but that this was already proven and there was, therefore, no necessity

to investigate the possibility of a psychogenic etiology.

Denial of the possibility of a psychogenic etiology in schizophrenia involves questions of both why this occurs and how it may be supported by research procedures. The most significant reason for the denial is surely the need to deny the unconscious with all its implications. Scheflen,[5] in his recent article, has very skillfully demonstrated many areas of operation of this need. The emphasis here is on the distortions in scientific method which resulted from this denial.

The experimental design involved the investigation and treatment of several hundred patients a year, and included intensive medical, dental, surgical and bacteriological procedures. While providing the patient with many hours of staff attention, this structure eliminated the possibility that any meaningful understanding of the patient or his life situation would occur. In other words the design restricted the inflow of information to the one preselected channel which in turn enhanced the apparent importance of those data.

Where data are obtained through another discipline, their significance may be further unwittingly distorted by specialists who, unable to understand general psychiatric theory, may nevertheless hold very strong psychiatric opinions. One of the criticisms made of Cotton, in 1922, was that he "wanted to throw over the data of organized psychiatry." Cotton in turn was much influenced by Frank Billings and E. C. Rosenow on whom he relied for much of his theoretical bacteriologic background. The accuracy of the criticism is confirmed by these quotations from a 1955 article by Rosenow:[6]

"The results of previous bacteriological and immunological studies on the etiology of schizophrenia and related mental disorders have been corroborated and extended in this further study, indicating that these conditions are due to a specific type of non-hemolytic, neurotropic streptococcal infection or intoxication.

"It is realized how contrary to current psychiatric tenets the concept that schizophrenia and related mental disorders can possibly be due to an infectious process, but the results of bacteriological studies by the special methods used indicate that such is nevertheless the case."

Without commenting on the validity of the bacteriologic data attention should be focused on the use which is made of subsidiary procedures in the investigation as a whole. The assumption is made that if one part of the investigation is conducted with a valid and proven scientific methodology then the whole is. Thus, accounts concerning the identification of strains of bacteria, pages of tables, descriptions of vaccines and surgical technics are used to give the impression of a methodology far more scientifically sound than it was in reality.

A process which might be called isolation can also be clearly seen in operation. When the initial efforts appeared to be resulting in success the failures were reconsidered and increasingly radical methods were applied with apparent further responses. When the most radical procedures had failed to produce recoveries, the failures were attributed to the results of too long standing disease processes. Critics were then challenged to repeat the procedures. Involved here was the denial that an extreme position had been taken, and that those who objected to severely mutilating operations performed on the basis of scant evidence would be unwilling to repeat the procedures. Thus, only those who held the same strong preconvictions would repeat their work. Great credit should be given to those who ultimately did repeat some of this work to disprove it.[7,8]

At the present time those who recommend performing a lobotomy during the first year of hospitalization for a schizophrenic illness place themselves in the same position.

A further example is the following quotation from a recent symposium on the insulin treatment of schizophrenia.[9]

"With our last 250 patients we have succeeded in freeing the 'Prozess-Symptome' so that the actual rate of remissions is now 100%. It is only on reaching this figure of 100% (and this percentage only) that further conclusions can be drawn, and that statistics may be produced on the later courses of schizophrenic patients treated with Insulin Shock. If this rate of remissions is not reached, the reason may be either false indications or incorrect treatment."

The dramatic discoveries and immunization procedures of the preceding years had created vistas where millions of people could be treated—or diseases prevented—by virtually magical methods. There must obviously have been a great desire to believe that the same dramatic results could be achieved in psychiatry. In effect then, there was a strong bias in favor of any theory or procedure which could put the cure of schizophrenia on a mass production basis. As was obvious in these studies the efficiency of a mass production treatment is then supported by maintaining sufficient distance from the patient so that arbitrary and meaningless criteria of cure can be used.[10] Or said in another way, it is usually easier for one doctor to "cure" 100 patients suffering from schizophrenia than it is for him to cure two in the same unit of time.

The inherent right of the physician to investigate carried with it the corollary that the setting was one in which it was possible to investigate etiologic factors. Facilities were created at numerous hospitals to investigate and treat the bacteriologic status of mental patients, which was valid investigation. The distortion which then occurred was the belief that without adequate other facilities, staff or controls, the investigation of the bacteriologic status was the investigation of etiologic factors in schizophrenia. There is obviously great personal resistance to acknowledging that one does not have adequate facilities to investigate etiologic concepts.

Adelaide Johnson and at least 13 co-workers at the Mayo Clinic have reported on the investigation of psychogenic factors in 27 schizophrenic patients.[11] A total of 64 relatives of these patients were seen, many in intensive psychotherapy. It required many months therapy with some relatives before they revealed information about highly traumatic events in the patients' lives which could have been obtained from no other source. If procedures of this order are necessary to obtain adequate data on psychogenic factors, then investigations lacking these facilities must of necessity be biased in their structural design.

Conclusions

A brief review has been made of some of the factors contributing to the belief that focal infection was the cause of schizophrenia. It has been suggested that occurrences in the recently preceding history of medicine and psychiatry made it almost inevitable that focal infection would be implicated as a cause of schizophrenia, and that some psychiatrists would wish to believe this. It was suggested that there was a continuum between the unconscious needs of some of the profession and the investigators which facilitated the acceptance of this theory.

Some of the needs causing distortion of scientific method were mentioned. The most prominent of these compulsive needs were attempts to deny the existence of the unconscious, feelings of guilt and inadequacy in relation to schizophrenic patients, omnipotent strivings, the need to maintain the concept of the physician as investigator in adverse circumstances, and the need to fill more adequately the role of healer.

Some of the operations through which distortion occurred have been outlined, and the manner in which these investigators isolated themselves from criticism has been described. It is suggested that there is a strong bias toward seeking magical cures. It is suggested that there is great need for truly objective investigations which may initially offer little prospect of helping any significant numbers of patients. Any attempt at finding a quick cure for schizophrenia, which ignores the particular individuality of the patient, may have its motivation in our unconscious needs and may, by the processes described, lead to similar distortions and excesses.

It was exactly 50 years ago, and over ten years before these investigations, that Adolph Meyer[12] stated "For all I can see, the main obstacle to a wider acceptance of a functional theory in terms of habit and complex conflicts and definite responses thereto is on the one hand the habitual or intentional lack of the necessary penetration into the life of the patient and family, and on the other hand the readiness of the physician to turn to set interpretations and to reiterate authoritative statements with a certain pedagogical self-sufficiency."

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CASE REPORT

Paroxysmal Nocturnal Hemoglobinuria: Its Place in Chronic Anemias

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PAROXYSMAL NOCTURNAL HEMOGLOBINURIA, or Marchiafava-Micheli syndrome, is a type of chronic hemolytic anemia known since about 1928.[1,2] This anemia develops because of the peculiar manifestation of hemolysis during sleep, and is characterized by chronic, normochromic, normocytic anemia, hemoglobinemia, hemoglobinuria and homosiderinuria.

The presentation of this case is to emphasize that, even though this disease is not common, it and similar anemias can readily be recognized by adequate history and appropriate laboratory examinations which do not entail elaborate and expensive procedures. The other point which this case emphasizes is that splenectomy has failed to show any lasting beneficial effect on the anemia or the hemolytic process.

Case Report

Mr. L. D. S. (M. H. No. 74630), a 38 year old white man, was initially seen on April 23, 1954, (at age 32), for an annual physical examination. His only complaints were sinus trouble, for approximately one year, a pilonidal cyst which drained periodically since 1952, and small verrucae on his back. Otherwise he was in good health; the physical examination was essentially negative except the pilonidal cyst, mild obesity and a questionably palpable spleen. The B.P. was 134/78. Laboratory work revealed a normal urinalysis, negative S.T.S., Hgb. 12 Gm., RBC. count of 4,400,000, WBC. count of 7,200 with a normal differential picture (Table 1). The sedimentation rate was 22 mm. The van den Bergh test was negative in the direct and 1.55 mg. in the indirect fraction. X-ray studies of the sinuses and chest were normal as was the EKG. Because of the slight elevation in the van den Bergh reaction, he was seen again in July 1954. It was learned that prior to his first visit there had been several persons with jaundice in his locale, and since the van den Bergh was normal on his return visit, it was thought that he had had a very mild, transient episode of hepatitis.

He was seen for his annual physical examination one year later, in April 1955, and other than an occasional episode of sinusitis had been in good health. Again the physical examination was unremarkable; the B.P. was 138/90-88. He was still moderately obese and at this time the Hgb. was 10.5 Gm., RBC. 35 million, with a WBC. of 4,800, and a differential count of 50% lymphs., 1% monos., and 49% segs. The sedimentation rate was 45 mm. Urinalysis revealed a trace of albumin. The van den Bergh test again showed a negative direct but the indirect fraction was 1.9 mg. per 100 ml. A fragility test showed beginning hemolysis at 0.42% and was complete at 0.34 per cent. In June 1955 he was seen again without any clinical evidence of jaundice but complaining of some fatigue. His Hgb. had returned to 12.4 Gm., WBC. count was 5,100 with lymphs. still about 41 per cent. Sedimentation rate had increased to 28 mm.; the indirect van den Bergh was elevated to 1.7 mg. per 100 ml. The total known protein was 5.95, albumin 4.95 and globulin 1.41 Gm. per 100 ml. Liver function studies were normal. No abnormal physical findings were noted. He returned in December 1955, asymptomatic, but the spleen was palpable two fingerbreadths below the left costal margin and was firm. At this time, it was suspected that the patient had *a hemolytic anemia, type undetermined.*

In July 1956, he reported that during the winter he had had bilateral frontal headaches, more noticeable in the morning, which he again attributed to sinusitis. There was a suspicion of dark urine and jaundice during the interval. Again the physical examination showed the spleen to be two fingerbreadths below the left costal border. He still had the pilonidal cyst and his blood pressure was 136/86. On this visit, the Hgb. was 9.55 Gm., RBC. 3.1 million, with a leukopenia of 3,200, WBC., with lymphs. 43%, monos. 2%, 1% immature forms with 54% segs. The sedimentation rate was 33 mm. per hour (Westergren). The urinalysis revealed a trace of albumin, tests bile and urobilinogen were positive, and there was an occasional hyaline cast. The van den Bergh test was normal and another fragility test showed the same results as the first one. Total serum protein and

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TABLE 1
LABORATORY STUDIES

	April 23, 1954	*April 1955*	*June 1955*	*December 1955*	*July 14, 1956*	*July 25, 1956*	*May 1957*	*July 1957*	*June 1959*
Hgb.	12.0	10.5	12.4	12.0	9.55	12.4	9.9	12.6	10.8
WBC.	7,200	4,800	5,100		3,200	4,400	7,200	6,600	7,000
Lymphocytes		50%	41%		43%	18%			30%
Neutrophils		49%	59%		54%	68%			62%
Others		1%			3%	14%			8%
Sed. rate mm./hr.	22		28		33		42		26
van den Bergh (mg.%)									
Total	1.55	1.90	1.70	0.85	0.30	0.26	0.25		
Direct	0.00	0.00	0.00	0.40	0.20	0.06	0.00		
Indirect	1.55	1.90	1.70	0.45	0.10	0.20	0.25		
Fragility		0.42 to 0.32			0.42 to 0.32	Sl. shift to left			0.38 to 0.24
Reticulocytes						10%		8%	2.4%
Platelets	Normal	Normal	Normal		Normal	141,200		495,500	350,000

Miscellaneous tests performed in June 1959:

Ham test	strongly positive
Cr^{51} turnover	9.5 days
Serum electrophoresis	normal pattern
Donath-Landsteiner	negative
Cold agglutination	negative
Lupus erythematosus preparations	negative

A/G ratio were normal, except for a slight elevation of the total protein to 8.2 Gm. per 100 ml.

He was seen in consultation at another hospital in July 1956, where it was noted that the spleen was down to 7 cm. below the left costal margin and there was slight enlargement of the liver. The findings were essentially the same as noted previously, with the addition of a 10% reticulocyte count. The bone marrow showed a typical picture of compensatory erythroid hyperplasia. There was no abnormal granulopoiesis. BUN. was 22 mg. per 100 ml. Liver function studies were normal except for thymol turbidity of 10 Maclagan units; the van den Bergh values were elevated with the indirect 2.0 and the direct 0.6 units. B.P. was 140/70. Gastrointestinal series was normal. On Aug. 6, 1956, a splenectomy was performed. The postoperative course was uncomplicated. The platelet count rose to 1,888,000, the reticulocyte count being elevated to 6.8 per cent the WBC. count rose to 7,900. The diagnosis at this time was *congenital hemolytic icterus with neutropenia and thrombocytopenia.*

He was next seen about 10 months later, in May 1957. He had no complaints; the B.P. was normal. The liver edge was palpable on deep inspiration and slightly tender. The Hgb. had decreased to 9.9 Gm., the WBC. blood count was 7,200, platelet count was 495,520, and there were 8% reticulocytes. He was started on Hematinic plastules, plain t.i.d. It was thought by one consultant that the picture represented further hemolysis, and it was then thought he might have the acquired type of hemolytic anemia.

Following this visit and during the remainder of 1957, the patient became more aware that the urine was darker in color, which he described as a "cocoa" or "Coca-Cola" in appearance; this was noted especially in August, September and November, accompanied by generalized malaise and fatigue. He was also aware that the urine was of this color after arising in the morning. He had transient periods of nausea and vomiting, with some slight increase in temperature.

In January, iron was discontinued, but because of a decrease in hemoglobin, was resumed after 3 months. During 1958, he had episodes of hemolysis in May; it was learned in retrospect that he had been aware of slightly colored urine after splenectomy. He recalled that he averaged two such episodes per month following splenectomy in 1956, but could not be sure that they were more frequent during the winter months. The physical examination on June 24, 1958, showed the B.P. to be 140/104-96; the liver edge was palpable on deep inspiration. He was essentially asymptomatic. It was thought that he was having very mild episodes of hemolysis secondary to an accessory spleen. However, he was continued on ferrous sulfate, 0.5 Gm. t.i.d.; the Hgb. averaged about 10.0 Gm.

In June 1959, he was admitted to Charleston Memorial Hospital for further investigation. Laboratory findings were as follows: Hgb. 10.8 Gm., PCV. 32 volumes %, RBC. count 3.5 million with a MCV. of 91 cubic microns (80-94), MCHC. 33.7% (30%), MCH. 30.9 micromicrograms (27-31). The WBC. count was 5,600 with 57% segs., 41% lymphs., 1% eos. and 1% basophiles. The urinalysis was negative except for a strongly positive iron reaction. Uroporphyrin and porphobilinogen were negative. BUN. was 11 mg. %. Platelet count was 350,000; reticulocyte count was 2.4 per cent. Coomb's test was negative and the fragility test showed hemolysis beginning at 0.38%, completed at 0.24% (control normal 0.45-0.36%). The blood was negative for the L.E. phenomenon. Electrophoresis of serum showed the following percentage distribution: albumin 62%, alpha I 4%, alpha II 4%, beta 15% and gamma 15 per cent. The Ham-Crosby test was positive and Cr^{51} labeled red cells showed a half-life of 9.5 days. The serum iron was 202 micrograms per 100 ml., Peters method (normal 56-183 micrograms %) and a bone marrow smear showed erythrocytic hyperplasia with occasional, bizarre, giant cells noted. Most of the cells appeared normocytic and normochromic.

Discussion

When a patient is found to have anemia,

it behooves the physician to investigate every possible aspect in an attempt to make an exact etiologic diagnosis. Even though the causes of anemia are numerous, the physician can make an accurate diagnosis in the majority without much difficulty by using the morphologic classification[3] as a guide. Thus, he may first determine whether the anemia is of the normocytic or macrocytic variety. The macrocytic anemias are related to either a deficiency of vitamin B_{12}, folic acid (directly or indirectly), or a reflection of intense activity of the bone marrow. Most of the anemias encountered clinically are of the normocytic variety, and microcytic anemia may actually represent a normocytic anemia of long duration or conditions related to deficiency such as a deficiency of copper or vitamin B_6. With the patient presented herein, it was noted that his indices were as follows: MCV. 91 cubic microns, MCHC. 33.7%, MCH. 30.9 micromicrograms, findings consistent with a normocytic anemia.

The first step in classifying any anemia is an adequate history and physical examination. The history of this patient was vague initially, but as his course progressed, he manifested the typical finding of paroxysmal nocturnal hemoglobinuria, e.g., hemolytic episodes occurring during sleep.[1-4] His age, absence of a family history of anemia, lack of evidence of blood loss and absence of exposure to hematopoietic toxins excluded other normocytic anemias. The other paroxysmal hemoglobinopathies, such as that associated with syphilis, cold hemoglobinemia and March hemoglobinemia are readily separated by the history, physical findings and appropriate laboratory studies such as the negative serologic test for syphilis, Donath-Landsteiner and cold agglutination test.[5] Otherwise, there is nothing to separate paroxysmal nocturnal hemoglobinuria from other types of anemia. After determining the morphology, the etiologic classification is the next step. The normocytic anemias may result from sudden loss of blood, destruction of red blood cells or lack of blood formation. The symptoms of anemia parallel the severity and rapidity of its development and it is not unusual to have an anemia of 8 to 10 grams without symptoms. However, there are usually some complaints in patients suffering from hemolytic states which are relieved when the anemia is corrected. This is especially true when symptoms are related to crises, such as abdominal pain, back pain, arthralgia, myalgia, and chills and fever. Such patients, however, may suffer from hemolytic states without any appreciable anemia if the compensatory activity of the bone marrow is able to keep up with the rapidity and chronicity of the hemolytic process. Crosby[6] and others have pointed out that in paroxysmal nocturnal hemoglobinuria, bouts of venous thrombosis in the extremities, the brain, portal system and kidneys may occur, and symptoms may then develop attributable to the areas of involvement.

There are no characteristic physical findings in hemolytic anemias, the degree of pallor and/or jaundice paralleling the degree of anemia. Splenomegaly is encountered in practically all hemolytic anemias and paroxysmal nocturnal hemoglobinuria is no exception. Bronzing of the skin may occur as noted in the patient presented, and hepatomegaly has also been reported. Generally, purpura is not a prominent feature of intracorpuscular defects but may be a noticeable phenomenon in extracorpuscular disorders, or if there is secondary hypersplenism from the associated splenomegaly that develops with the hemolytic anemias.

The degree of anemia in paroxysmal nocturnal hemoglobinuria may be relatively mild or, occasionally with crises, may be very severe. The major manifestation is chronicity with an associated iron deficiency type anemia. Ham[1] pointed out that when the plasma hemoglobin is greater than 30 mg. per 100 cc. hemoglobinemia occurs, and when hemosiderinuria is noted the iron is a reflection of the hemoglobinuria. Leukopenia and thrombocytopenia can also occur, as was found prior to splenectomy in our patient. The finding of thrombocytopenia without splenomegaly would be rare indeed with intracorpuscular hemolytic anemia. The bone marrow examination is not generally diagnostic except for the compensatory reactive erythroid hyperplasia, which is consistent with any hemolytic process though aplasia has also been encountered.[7,8] There is no abnormality in the megakarocytes, and in examinations as plasma and hemoglobin

electrophoresis, Coomb's test, fragility test, Donath-Landsteiner test, serologic test for syphilis and coagulation studies are normal.[2,5,7-9] In 1939, Ham[1] reviewed his studies on destruction of red blood cells and described the hemolysis test with acidified serum. He found that with paroxysmal nocturnal hemoglobinuria, the patient's cells would not hemolyze in acidified normal serum. Our patient showed a typical positive Ham test, which actually enabled us to make the positive diagnosis of paroxysmal nocturnal hemoglobinuria.

The etiology of paroxysmal noctural hemoglobinuria has not been established, even though heredity, nutritional deficiencies, endocrine dysfunction, neoplastic disease and infection have been suggested as factors. These have never been consistently positive in patients with the disease.[2,5,6] Crosby[4] has pointed out that such patients probably represent a "constitutional predisposition" for this type of anemia. Even though the etiology has not been discovered, there has been a great deal of investigation of the pathophysiology and the relation of the hemolytic process to sleep. It is generally accepted that the defect during sleep, either day or night, is due to the slight acidification of the plasma, which, in turn, alters the apparently abnormal[8-10] stromal protein of the red cells which may be related to some metabolic defect in the red cell itself.

After one makes the diagnosis of paroxysmal nocturnal hemoglobinuria, the question of treatment arises. The acceptable therapy now is to eliminate harmful procedures rather than to embark on a positive treatment program. Practically every form of treatment has been found to be unsuccessful or harmful except for the administration of iron. Methods have been used to counteract the changing pH defect, all without lasting benefit and frequently associated with increased hemolysis when the basic substance is withdrawn.[1,11,12] Ham[1] postulated sodium bicarbonate as the agent of choice, but noted an increased number of hemolytic reactions when this agent was discontinued. Splenectomy has been of no benefit and is not infrequently accompanied by increased hemolysis, not only postoperatively but may occur during the operation or develop again during the course of the disease. This is in contrast to congenital hemolytic anemia and in certain secondary states of hypersplenism, in which splenectomy is almost always of benefit. The use of transfusions has been associated with increased frequency of hemolysis; it is recommended that when administration of blood is necessary, washed red blood cells should be used. The big objection to transfusion is the possibility of producing iso-antibodies, thus make necessary transfusions hazardous.

Various anticoagulants have been used or advocated with episodes of vascular occlusion. Williams[13] investigated the use of alpha-tocopherol phosphate but found it was of no value in protecting the patient against thrombotic episodes. Dicumarol has also been used in the treatment of thrombotic episodes but has not gained widespread acceptance either as a prophylactic measure or during hemolytic crises. Heparin, on the other hand, increases the activity of the clotting system in paroxysmal nocturnal hemoglobinemia, probably blocking the two inhibitors of hemolysis which Crosby has noted as being present in the serum.[4,8] Therefore, this agent should not be used in patients with paroxysmal nocturnal hemoglobinuria. Since large amounts of iron are lost in the urine, there is a corresponding need of increased intake of iron. This is one hemolytic disorder in which iron therapy is necessary and, as was noted in our patient, the hemoglobin could be maintained at adequate levels on ferrous sulfate, 0.5 to 1 Gm. daily. The administration of hormones and steroids has been tried without benefit, and vitamins and minerals, with the exception of iron, have shown no lasting benefit. There is no guiding factor concerning the patient's prognosis. Crosby[6] has noted survival up to 35 years but, generally, if the manifestations of thrombosis, either renal, cerebral or portal, develop the prognosis is not good. Hypertension is not an unusual manifestation in paroxysmal nocturnal hemoglobinuria, even though the exact relationship has not been correlated with the disease. We wish to emphasize that though our patient had anemia almost continuously for five years, he has been able to lead a normal, productive life, without any appreciable loss of time from

work due to illness, except for periods of hospitalization for splenectomy and diagnostic studies.

Summary

1. A patient with paroxysmal nocturnal hemoglobinuria is presented to emphasize the importance of an adequate history, physical examination and appropriate laboratory studies in the diagnosis of anemia, especially of this type.

2. Paroxysmal nocturnal hemoglobinuria produces a chronic normocytic anemia, of unknown etiology, though it is thought to represent an intracorpuscular defect, although extracorpuscular alterations are necessary before hemolysis develops, such as sleep and/or change in the pH of the plasma.

3. The laboratory findings of significance will be revealed in the complete blood count, bone marrow examination, urinalysis, particularly for bile, iron, free hemoglobin and red cells, reticulocyte count and the Ham test. Other hematologic tests which are usually normal are, the fragility test, Coomb's test, plasma and hemoglobin electrophoresis. Radioactive chromium tagged RBC. and ferrokynetics, while not diagnostic in themselves, will provide useful information in the study of patients with hemolytic anemias.

4. Treatment of paroxysmal nocturnal hemoglobinuria is predominantly replacement of iron, transfusions of washed red blood cells when necessary, and avoidance of splenectomy. At times it may be necessary to use anticoagulants (coumarin derivatives) if hemolytic crises are accompanied by thrombosis, but not heparin. The prognosis is generally fair, especially in patients who do not manifest the major complications of thrombosis or severe hemolytic crises.

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The Emotionally Disturbed Child: Problems Within the Family*

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That the development of a child is influenced by family environment is an unquestioned fact. The management of an unwholesome situation is an extremely difficult one. As the author shows, it is not as simple as removing the child from his environment. Treatment of the family situation is basic, if feasible.

Part I

FAMILY LIFE and its relation to mental illness has been studied in two ways. One is the position of the family in the larger culture and the changes in family life as a result of changes in culture. Many excellent studies have come from the sociologists and anthropologists, works such as Hollingshead and Redlich's[1] *Social Class and Mental Illness* and Faris and Dunham's[2] *Mental Disorders in Urban Areas.* Works which do not deal directly with the problem of mental illness but in a more general sense with the problems of adjustment in present-day culture are the writings of Reisman[3] and of Seeley.[4] There are also studies of particular types of maladjustment such as delinquency and crime and their relation to family life in various cultures.

The findings and the theories expressed in these books are valuable to our full understanding of the problem of mental illness and family life. There are changes in society as a whole which make stable and peaceful family life quite difficult to maintain. The social structure does not, as formerly, surround the family with many supports, such as close ties of kinship and a universally accepted code of conduct. The family has become an isolated conjugal unit. The goal of families in our economy is seldom that of mere survival, but of higher social and financial position. The strivings toward these goals are accompanied by considerable anxiety and often by a lack of integration between economic activity and peaceful family life. One of the contributions of anthropology has been the study of various cultures to find the range within which the human organism can adapt to cultural conditions.

The second approach is the clinical study of the emotionally disturbed individual and the relation of his illness to the family environment. The psychiatrist who treats children has known that their emotional disorders are caused or aggravated by the nature of their relationships to other members of the family. This concept has been accepted from the beginning of modern psychiatry, but it is only in recent years that the relationship of the child's illness to the emotional climate of his family has been intensively studied. The investigations on the effects of maternal deprivation first published less than 20 years ago, followed by the studies of the mother-child relationship in early infantile autism, appear to me to have been the beginning of this new awareness of the importance of family life.[5-7] Direct observation of infants and young children, such as those studied in the Hampstead Nurseries by Anna Freud[8] confirmed, elaborated and revised the knowledge concerning early mother-child relationships which had been formulated from the material learned from analyses of adults.

The child psychiatrist and the social worker working in the guidance clinic and the family casework agency have long known that the improvement of emotionally ill children depends in a large measure upon improvement in the family relationships.

This paper, then, deals with the diagnosis and treatment of the child, not as an isolated

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individual, but as a member of a family. The term "emotionally disturbed" includes not only those maladjusted children who are intellectually and physically normal, but also the mentally retarded, "brain damaged" and physically handicapped. For the handicapped child there is the problem of his own and his family's adjustment to the handicap. Parental rejection, overprotection and other distortions in parent-child relationships can effect the mentally and physically handicapped child. Many children who are diagnosed "mentally retarded" might better be called "socially retarded," because the stresses of family life do not permit them to take advantage of their modest intellectual endowment. The child who is blind or crippled may never compensate for his affliction because of feelings of unworthiness stemming from parental rejection.

There are many historical details which the clinician must know to diagnose the child's illness in relation to his family life. I will discuss briefly two important sources of information. One of these is the developmental history of the child; the other is what I shall call the "nurturing" behavior of his parents.

Part II

The unfolding of *the child's personality* proceeds in such an orderly fashion that we can describe several distinct stages of development. In each stage the child has certain needs and requires certain relations with other persons. If the needs of any stage are not satisfied, growth through later stages is hindered or distorted.

(1) *Stage of Intra-uterine Life.* The needs of the child are mainly physical. There are studies in progress which *hint* that the parental attitude may affect the physical environment of the fetus. Although the attitudes of the parents cannot directly affect the child at this time, parental attitudes toward this child may influence later development. (The historical fragments which I will cite throughout the remainder of the paper concern children who are seriously emotionally disturbed.)

Mrs. C. was extremely upset about her pregnancy. Her husband was in the Marine Corps and she felt that the pregnancy was too much of a responsibility for her. Each time he would get leave she would create a scene upon his leaving, pleading with him to stay. Mr. C. said that at first he looked forward to these leaves from military duty, but these parting scenes upset him so that he felt reluctant to visit his wife.

Miss B.'s mother was a cruel person who looked upon all sexuality as sinful. She forbade her daughter to associate with men because "all men had only one idea." Miss B. became pregnant and her mother told her that the unborn child was a product of sin and doomed to be bad.

The discomforts and disfigurements of pregnancy, the fears and pains of childbirth, the economic state of the family and many other factors can be important during this stage.

(2) *Stage of Neonatal Life.* During these early months the infant's basic physical needs must be satisfied. He is powerless to assuage the pangs of hunger, to protect himself from pain and from frightening noise. The giving of food is the beginning of a relationship to the giver of food. The child who is not satisfied at this stage will always have anxiety.

John's mother did not want to care for him after his birth so she left him with her mother. But her mother did not wish to care for John so she left him in the care of an illegitimate teenager who was boarding in her home. This girl left the child in his crib all day, seldom changed his clothes and fed him irregularly.

Peter's mother had a moderately severe postpartum depression but was not treated. Very often Peter would cry for hours from hunger but this mother could not summon the energy to feed him. Peter's father noticed nothing abnormal about his wife or son during these months, said that taking care of babies was the woman's job.

(3) *Stage of Primary Identification.* During this time the child reaches out to form a strong union with his mother. The strength of the child's personality and his development from the helpless infant of the neonatal stage depend upon this union. A lack of this union produces panic and withdrawal. This union Bowlby calls a "love relationship," an apt phrase, for without this union the child seems incapable of developing strong ties of affection later in life. It is in this stage that the effects of maternal deprivation are so severe. This syndrome of "maternal deprivation" was first recognized by Lowrey,[6] Goldfarb[5] and Spitz[7] in children who lived in orphanages. But maternal deprivation can occur in the parental home, in foster homes, and can produce not only the "affectionless" child but has a part in the genesis of psychotic illnesses of childhood.

(4) *Stage of Individuation.* The child begins to see himself as an independent creature and begins to reach out for relationships with others. Here begins the great struggle to control his impulses and to learn to tolerate

anxiety and to postpone the need for immediate satisfaction of drives. The defiance, negativism, and aggression which become manifest at this stage become a real test of parents. Do they look upon the child's impulse life as bad? Do they wish to keep him an infant? Do they think all of this is cute? Are they harsh, or do they make no effort to train him? Are they worried about what grandmother or the neighbors will say about their abilities as parents?

Anthony T. was a very active child and very early showed a negativistic attitude toward food. Mrs. T. attempted to force feed him, then to bribe him, to punish him. Every device was tried over and over again. His negativism spread to sleeping habits, to refusal to be bathed, to almost everything.

(5) *Stage of Sexual Differentiation.* As the child becomes aware of sex differences, there is an awareness of himself as belonging to one sex. The child of each sex makes an identification of a distinct type to each parent. When there is only one parent, when there is severe conflict between parents, rejection of the child in favor of a younger child, unusual indulgence or unusual frustration of the child, he will have difficulty in resolving his conflicting feelings about his parents. On the outcome of this phase will depend his attitude toward his siblings with whom he must share his parents, his attitudes toward persons of his own sex and of the opposite sex. We have many examples of emotionally disturbed children whose fathers or mothers deserted during this period, who were exposed to scenes of adult sexual relations, or whose fathers were harsh.

(6) *Stage of Extrafamilial Development.* During this period the child moves to interact with more persons outside of the family, he begins to test social reality. It is at this time, if the child has received the satisfactions he needed in earlier periods, that two very important things happen. His energies, which until this time have been occupied with controlling his instinctual life and with establishing his relationships to his parents, are now freed for learning. Since he has made healthy identifications with his parents he is able to accept social realities, standards of honesty and fair play, and other authority figures as he accepted his parents. If development has not proceeded smoothly he will have learning difficulties and problems with authority. It is no accident that most of the behavior disorders do not come to attention until the child has entered school. And it becomes understandable that many parents should blame the school because the faulty development of personality was not evident to them before school started.

(7) *Stage of Adolescence.* The intensification of sexual impulses disturbs the equilibrium of the person, and the wide swings in mood and behavior so characteristic of adolescence are attempts to restore this equilibrium. If the equilibrium between the instincts and the ego has been unstable and unhealthy, old conflicts will be stirred up, acting out and emotional liability will be intensified. It is said that puberty is a repetition of the infantile sexual period and a repetition of the infantile conflicts the child had regarding his parents. It is a final breaking away from infantile ties with parents so the individual can make a mature relationship in marriage.

In all of these stages there is almost an infinite variety of interactions between parents and child. Not all of the faulty attitudes of parents will produce disastrous results in the characters of their children, and we sometimes see children who are surprisingly intact despite severe rejection and deprivation. We also see children whose personalities appear so fragile that relatively insignificant trauma produces serious maladjustment. The mentally retarded or physically handicapped child may need to linger longer in each stage, but there is the danger that protection beyond what is needed for his particular disability will hinder the development of his personality. It is often difficult to decide where appropriate parental indulgence ends and overprotection begins.

Part III

The second important area of our clinical investigation I choose to call the *"nurturing" behavior of parents.* I use this term because it is often difficult to distinguish clearly between "mothering" and "fathering." But we can speak of a relation of members of the family to one another which is conducive to the healthy development of the family unit. This nurture can exist even when the parental couple are neurotic persons, for the marriage is not simply the sum of the personalities of the two partners. Neurotic individuals, despite traumatic childhood, often strike a balance and become good parents. Therapy for the individual husband or wife who is neurotic does not necessarily improve a poor marriage. Although there is more likelihood of neurotic people

forming unhappy unions, we must look for more than the presence or absence of neurosis. A stable marriage will result if the partners are able to offer mutual nurture, that is, if they are able to satisfy one another's needs, can give support for personal identity and worth, and can strengthen defenses against anxiety. When there is a failure of this support, a failure in sharing pleasure, a failure in sexual relations, in sharing of responsibility and authority, there will be faulty motivation for having children and faulty rearing of children.

The nurturing behavior of the mother, certainly in the stages of intra-uterine life, neonatal life, and the stage of primary identification, is mainly biologically oriented, and the nurture which the father supplies is to the mother-child unit rather than directly to the child. When the child reaches the stage of individuation he recognizes his father and from that time both parents begin to have an equal importance for the child. When there is a "disturbance in mothering" the father contributes to this if he has failed to give the protection and emotional support which enables the mother to feel adequate in her role.

The child can play a variety of roles in the lives and phantasies of his parents, and an examination of the attitudes and aspirations of the parents regarding their child can tell us much about the reasons for the child's disturbance. In the happy family the child will be an extension of the love the parents share and they will find in the child the qualities they treasure in one another. When the nurture is lacking and there is conflict between the parents, then hostility arises. This hostility can be displaced from the area of conflict between parents to conflict between parents and child. The child who is the target of this hostility feels rejected, rejected because he is "bad." When there is a sexual difficulty, the sexually unsatisfied parent can seek compensatory love from the child. This will lead to seduction of the child. When a father is insecure about his own masculinity he will view the awkward or retarded child as a threat and will need to deny the defect in this child, to push the child to unattainable goals. The parents who depend excessively upon the esteem of associates or their own parents will need to raise "good" children.

Parents can react in many ways to deviant behavior of their child. They can conclude that the child was born "bad" and they can do nothing about it. They can turn away from the problem and develop many outside interests to get away from the child. They can punish the child for their failure, or can project the blame onto the doctor or the teacher. They can submit entirely to the child's desires. They can accentuate the physical care of the child—overfeed him, worry about slight illnesses, all the while neglecting him emotionally. Because of hostile feelings toward their own parents, which they have hidden under excessive conformity, they derive satisfaction from the acting out of their child.

Part IV

If improvement of the family is necessary for the well-being of the disturbed child, what is the status of *family-centered treatment*? How can we provide for this within our accepted methods of treatment, or how can current methods be modified? For the most part treatment of the emotionally disturbed and mentally or physically handicapped child remains still the treatment of the individual child.

There is failure to use the knowledge of the family unit in the diagnosis and treatment of these children. The diagnostic terms of adult psychiatry, terms which connote a type of finality in personality structure, are used to describe the child whose personality structure is still developing. The concepts of normality and abnormality of behavior often used in adult psychiatry do not apply to children. The child cannot change and control his environment, he does not know the nature and consequences of his behavior as the adult does. Ackerman writes:

> ". . . child therapists are often impelled to undertake treatment of a child without a clearly defined clinical diagnosis and without a dynamic appraisal of the total functioning of the child's personality within the framework of a defined family environment. At the least, this is bad practice. How can one presume to correct what is wrong with the functioning of a child's personality without first establishing what is wrong? Too often, therapists treat without knowing what they treat."[9]

Distortions of the concepts of family dynamics hinder treatment. Parents are condemned. They have rejected the child, have exploited him, and have damaged him. Remove him from these bad parents and place him with good parents. But the mere act of removing a child from an unhealthy family does not cure

his illness; often it aggravates it. More ways must be used to help parents. In dealing with the families of these sick children social workers have had to modify one of their most cherished concepts, namely, that the client must assume the initiative in seeking help if treatment is to be successful.

"... In order to obtain any positive results with the seemingly hopeless group, we have had to take a fresh look at what casework has to offer and use all the ingenuity and skill at our command to adapt our service to its needs."[10]

There are significant differences among "rejecting" mothers and "punitive" fathers. We can think of all parents as spreading over a scale that ranges from those with a high capacity for understanding and accepting the changes which take place in their children and capable of making the necessary changes in themselves, to those parents who possess relatively little ability either for accepting the changes in their children or effecting changes in themselves. The practice has been to give help to those fortunate enough to be in the upper ranges of this scale and to remove the children from those in the lower ranges. When it is judged that the environment is extremely detrimental to the well-being of the child, and it is predicted that the potential of the family for change is so low that the child will continue to be ill no matter what treatment is given to him as an individual, it is then necessary to decide on his separation from the family. But this decision must be made only after all resources and skills have been used to mend the family. Bowlby has written:

"Faced with a situation where the co-operation of key people is difficult to maintain, there is a temptation for the professional worker to solve the group problem by removing one or more of the individuals concerned. In industry, management may wish to sack the trouble-maker. A similar procedure in child guidance has been to take the child out of the home and put him elsewhere. Although occasionally unavoidable, this seems to me a policy of despair."[11]

Placement of seriously disturbed children, neglected children, and often mentally retarded children in institutions hinders the treatment of family disease. The rejected child will see this as a further rejection, and some children so removed from their families never regain their psychologic place or even their physical place in the family. The community, the family, the child, and often the staff of the institution feel that the child is the cause of his disturbance. The community and the family hand to the institution all responsibility for treatment. These difficulties are often increased by institutional practices. Many institutions are isolated physically and operationally from the community and the family. Contacts with parents are discouraged. The parents have no opportunity to exercise parental authority and responsibility.

Bloch and co-workers,[12] in *A Study of Children Referred for Residential Treatment in New York State*, report that the disturbed children *and* their families were known "early and often," and there was much repetition of diagnostic services, but

"... they received diagnostic referral and placement services and very little else in the way of treatment, such as casework, psychotherapy, or special educational arrangements."

It was found that when these children were first known to an agency, 83% were in their own homes, but at a follow-up date only a few years later only 36% were in their own homes. More than half of the children came from "multiproblem" families.

We can speculate on the meaning of these figures. Certainly case finding is good; childhood emotional disturbances are being recognized early. Do these figures mean that where there is less severe disturbance in the family the child and family are helped and this is because the families are not "resistant" to help? Can we say that where there is severe family disease the child and the family are not helped and the child is eventually placed outside of the family? Does it mean that there are not enough caseworkers and psychiatrists to go around and the more rewarding cases and the more cooperative parents get what is available?

"... the large proportion of children who received no rehabilitative services; 72% had no psychotherapy, 85% no casework, and 71% no special school services; but 81% had been placed."[13]

The usefulness of any institution or other treatment facility depends not principally upon the amount of trouble the child causes his family or community, but on the degree to which he can be expected to integrate his experiences in treatment into his personality structure. Removal of a child from his family must only be made when there are definite indications, such as a need for an experience in corrective group living, for making better relations to authority figures, or for gaining mastery over impulses. This removal must be part of a plan for helping the family to find a

better balance and enabling the family to further mutual nurture.

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HOTEL RESERVATION APPLICATION FORM

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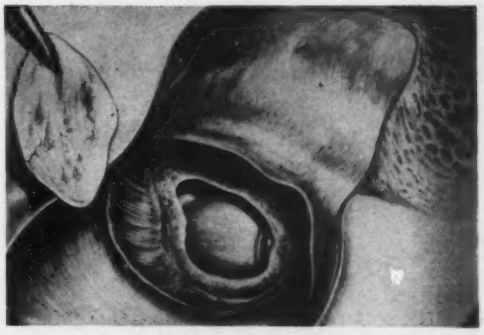
Mastoid Surgery in Chronic Otitis Media Without Creating a Mastoid Cavity*

JACK W. POU, M.D., *Shreveport, La.*

The author is applying newer surgical concepts to obtain better cosmetic results and to avoid morbidity in the surgical attack on chronic disease of the ear. The ultimate objective is conservation of hearing.

THE SURGICAL INDICATIONS for chronic otitis media were formerly considered to be the "dangerous" ear, with marginal perforations and cholesteatoma and far advanced disease of the middle ear with disruption of the ossicular chain, nonserviceable hearing and persistent drainage. Chronic disorders of the ear with central perforations in patients with serviceable or borderline hearing were treated conservatively to preserve any hearing that remained. To adhere to these indications for surgical treatment meant that most chronic diseases of the ear were not attacked surgically, and therefore the patients were left with a persistently or intermittently draining ear and some conductive hearing loss. The main reasons why this type of ear was not treated surgically were fear of making hearing worse and the undesirable postoperative mastoid cavity that it created (Fig. 1).

FIG. 1



Old type II tympanoplasty with skin graft and membranous canal wall folded back into mastoid cavity. This is not done now in order to avoid a postoperative mastoid cavity.

Newer Surgical Procedures

The many advances in otologic surgery during the last few years have greatly broadened the indications for surgical intervention in chronic diseases of the ear. With microsurgical technics the otologist can readily remove diseased tissue from the attic and off the ossicles without danger of disrupting the ossicular chain and causing increased hearing loss. However, many ears are still not operated upon because of the resulting prolonged postoperative care of the mastoid cavity.

A relatively new surgical approach to chronic diseases of the ear has been devised, in which a flap is not turned and thus a mastoid cavity is not created. It is called atticomastoidectomy. In April 1958, Henner and Jeantet[1] presented 9 cases in which this technic was used. I have performed this operation on 12 patients with chronic disorders of the ear with highly satisfactory results (Table 1). A dry ear was obtained in all 12 and closure of the perforation was obtained in all but three.

It should be emphasized that nonsurgical treatment should always be tried before surgical intervention. A fairly high proportion of chronically diseased ears will respond favorably to parenteral administration of antibiotics and the usual cleansing, cauterization of granulation tissue and insufflation of different specific powders. Adenoidectomy should be done when indicated and measures should be instituted to control any allergic, sinus or throat condition which could be a chronic source of involvement of the ear.

*Read before the Section on Ophthalmology and Otolaryngology, Southern Medical Association, Fifty-Third Annual Meeting, Atlanta, Ga., November 16-19, 1959.

TABLE 1

PERTINENT DATA ON 12 PATIENTS TREATED BY ATTICOMASTOIDECTOMY FOR CHRONIC DISEASE OF THE EAR

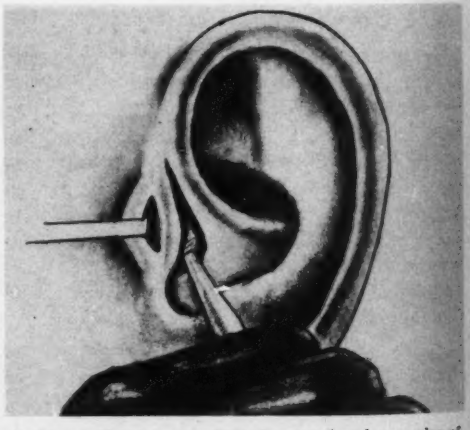
Patient	*Age Years*	*Duration Years*	*Perforation*	*X-ray Findings*	*Incision**	*Skin Graft*	*Dry Ear*	*Closure of Perforation*	*Hearing*
1	40	15	Small in area of stapes; cholesteatoma in attic	Sclerotic	End.	0	+	0	Improved
2	19	19	Large central	Sclerotic	PA.	+	+	+	Normal (rt. ear)
3	19	19	Large central	Sclerotic	PA.	+	+	+	Normal (lt. ear)
4	29	9	Small in area of stapes with polyp	Sclerotic with possible cavity in attic	End.	0	+	+	Normal
5	23	1½	Small central	Cloudiness of antral and peri-antral cells	End.	0	+	+	Normal
6	13	5	Small in area of stapes with polyp	Cloudiness of mastoid cells with evidence of cell destruction	End.	0	+	+	Improved
7	14	3	Large central	Cloudiness with evidence of active disease	End.	0	+	0	Unchanged
8	19	3	Small central	Haziness with evidence of active mastoid disease	End.	0	+	+	Normal
9	36	6	Pinpoint, with thick, profuse, gluey drainage	Cloudiness of all mastoid cells with evidence of cell destruction	End.	0	+	+	Unchanged; had superimposed nerve deafness
10	38	30	Small central in area of stapes; cholesteatoma in attic	Sclerotic	End.	0	+	+	Normal
11	12	10	Large central; cholesteatoma in attic	Sclerotic with possible cavity in attic	PA.	+	+	+	Normal
12	15	7	Large central	Sclerotic	PA.	+	+	0	40 DB

*Incision: End.—endaural; PA.—postauricular.

Atticomastoidectomy without turning a flap is indicated if an ear with a central perforation fails to respond satisfactorily to such conservative treatment, since the percentage of good results is high and the postoperative morbidity rate is negligible. This includes chronically diseased ears that particularly do not pose a hearing problem. Complications still can arise with an acute exacerbation from a central perforation in the absence of cholesteatoma. Because of the potential dangers of continued infection, because this constitutes a chronic focus of infection, and because of the socioeconomic disadvantages of continuous malodorous discharge operation is advisable.

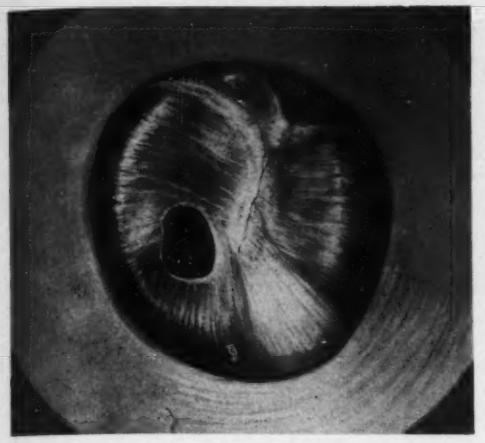
Atticomastoidectomy is done through a labyrinthectomy type of endaural incision (Fig. 2), if the perforation is small and a skin graft is not needed to obtain closure of the perforation (Fig. 3). This type of incision enables easy primary closure. A large perforation necessitates a skin graft for closure, and a postauricular incision should be used (Fig. 4). This permits an additional small anterior endaural incision which facilitates vision of the anterior margin of the perforation by means of a small self-retaining retractor and enables the surgeon to use both hands while doing the myringoplasty. If skin grafting is attempted through the endaural labyrinthectomy type in-

FIG. 2



Incision made more posteriorly than usual and no wedge of skin or subcutaneous tissue removed. This facilities easy closure.

FIG. 3

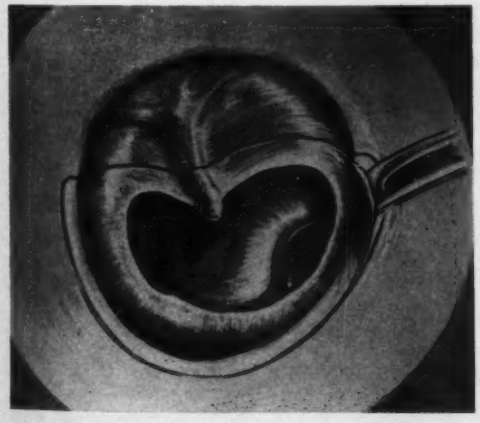


Perforations of this size and smaller will heal without grafting after subsidence of infection.

cision, vision of the tympanic membrane is the same as with an ear speculum without an incision and often is inadequate. If an additional anterior incision is made, it is extremely difficult to close both endaural incisions primarily.

Complete mastoidectomy is performed, including atticotomy, with careful skeletonization of the labyrinth, dural plate and ossicular chain. The posterior and superior bony canals are preserved. If the perforation extends up to the area of the stapes, the membranous canal wall is elevated to the annular rim and the middle ear is entered (Fig. 5). This provides the same exposure to the area of the stapes as

FIG. 4



Perforations this size always require skin graft. Primary grafting at the time of operation on the mastoid is usually successful.

FIG. 5

Exposure in stapes area obtained by elevating membranous canal wall and entering middle ear. The bridge can be removed to facilitate cleaning the incus and the footplate area.

in stapes mobilization. If granulation tissue extends up toward the attic surrounding the long process of the incus, the bony bridge is taken down, which provides visualization of the entire incus. Since a flap will not be turned, removing the bridge does not cause postoperative fixation of the ossicular chain.

The remaining part of the middle ear is cleansed through the perforation with curettes and a polishing burr. By this technic of cleaning the attic through the atticomastoidectomy approach, elevating the flap and cleaning the area around the stapes, and cleaning the middle ear through the existing perforation, it is possible to cleanse the ear in a manner fairly comparable to that in radical mastoidectomy but leave the ossicular chain and the tympanic membrane remnants intact. If the perforation is small, the edges should be freshened in an attempt to obtain primary closure. If closure does not occur spontaneously, the edges of the perforation should be cauterized at repeated intervals according to the technic of Wright.[2] If a skin graft is required, a thin, full thickness skin graft is placed over the perforation by the usual myringoplasty technic advocated by Wright.[2] The endaural or postauricular incision is closed primarily with a small rubber dam drain in the lower part of the incision. This is removed in one week when the sutures are removed.

In the 12 patients operated upon, the duration of the disease ranged from 1½ to 30 years. The size of the perforations ranged from small pinpoint to large ones involving almost the entire tympanic membrane. Roentgenographic

observations ranged from the completely sclerotic mastoid process to cloudiness and haziness of the mastoid cells with loss of contour and some cellular destruction. The postauricular approach was used in the 4 cases in which a skin graft of tympanic membrane was required; closure was obtained in three. Myringoplasty is planned for the 3 patients in whom the perforation failed to close if cauterization of the edges of the perforation proves unsuccessful.

The pathologic alteration observed at operation is usually a hyperplastic membrane lining the middle ear, attic and mastoid cells. Two patients had diffuse involvement of all mastoid cells with thick, gluey-like, serosanguineous drainage. The culture of this draining exudate was sterile. Three patients were found to have an isolated cholesteatoma in the attic, probably due to healed attic perforations.

Summary

Twelve patients, with suppurative otitis media and with central perforations which did not respond to conservative treatment, were treated by atticomastoidectomy without turning a flap, and thus avoiding creation of a postoperative mastoid cavity. The middle ear was cleansed of granulation tissue in the area of the stapes by elevating the flap and entering the middle ear. This provided exposure similar to that provided by stapes mobilization, which enables thorough cleansing of the posterior half of the middle ear. The remaining part of the middle ear was cleaned through the perforation. If granulation tissue extends up toward the attic, the facial bridge is taken down; this permits a more thorough cleansing of the ossicles. Since a flap is not turned, taking the bridge down does not interfere with ossicular function.

If the perforation is large enough to require a skin graft for closure, the atticomastoidectomy is done through a postauricular approach. The reason for this is that an additional anteriorly placed endaural incision can be made and a self-retaining retractor put in place, which allows the operator to use both hands for the myringoplasty. If the perforation is small, it will usually close spontaneously after infection has subsided.

A labyrinthectomy endaural type of incision is used for the atticomastoidectomy when the perforation is small and a skin graft is not required. This type of incision facilitates primary closure. A rubber dam drain is placed in the lower part of the incision and removed after one week.

A dry ear was obtained in all 12 patients and the perforations closed in all but three.

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Discussion (Abstract)

Dr. Harold G. Tabb, New Orleans, La. Doctor Pou has realized as many of us have that there are disadvantages to the present surgical procedures on the ear for eradication of disease which create a postoperative mastoid cavity. This defect is undesirable from a functional as well as a cosmetic standpoint and there is resentment in patients who must return after operation for repeated care of a cavity created during so-called "corrective" ear surgery.

This undesirable defect was common in the earlier types of radical mastoid surgery, and is still present in the newer types of tympanoplastic surgery which have become so popular recently and which have replaced earlier technics in most hands.

I am pleased to hear a technic described today that places emphasis on conservatism and strives to conserve anatomical structures and retain relatively normal function.

The trend of development for this type of surgery is a natural and evolutionary change made possible with our newly developed armamentarium for microscopic surgery which permits more thorough and exacting technics under the protection of the wide spectrum antibiotics.

We have been interested in this problem of eliminating the mastoid cavity for a long time particularly in children where these operative defects are such a handicap during their early developmental years. I am in thorough accord with the principles and objectives as suggested here today.

I have had some experience utilizing Dr. Pou's procedure of endaural atticotomy with or without postauricular mastoidectomy and have found it uniformly successful when infection is controlled and all squamous epithelium is removed from the middle ear and mastoid areas.

I have had some difficulty with this method in exposing the anterior tympanum, such as the eustachian tube orifice and the round window niche through the existing perforations unless these are extremely large.

And I have hesitated in removing more than a few millimeters of the distal segment of the posterior superior wall of the osseous canal even though no tympanomeatal flap is created, for fear of allowing adherence of meatal skin to the ossicles with postoperative fixation.

These are not great disadvantages, but in attempt-

ing to overcome them and to eliminate the postauricular incision, we have recently performed a series of similar operations endaurally using a stapes type incision with a large meatal flap for removal of disease of the middle ear and reconstruction of the sound conducting apparatus, and a deep endaural incision for the mastoid work.

The posterior wall of the canal was maintained at all times although thinned to a paper plate even if the ossicles were removed. This prevents a mastoid cavity.

We believe so strongly about preserving the canal wall that we are at present working on a method, as shown in this drawing (slide shown), of removing temporarily a segment of the posterior wall of the osseous canal for unobstructed work on the ossicles and facial ridge and then replacing the bone and fixing it in its initial position with Ostamer (bone glue).

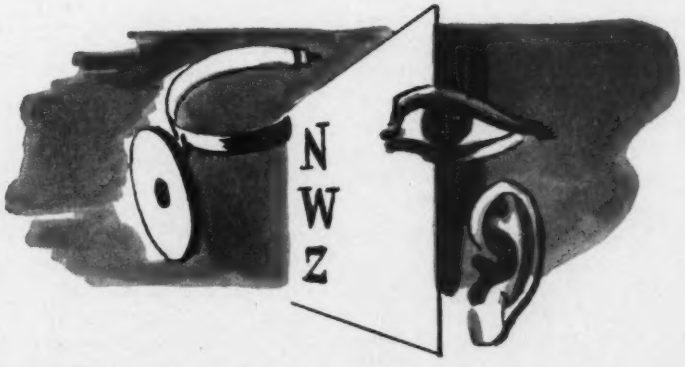
After elimination of disease, the endaural incision is closed primarily with absorbable sutures around a polyethylene drain from the mastoid cavity. Reconstruction of the sound conducting mechanism is performed through the stapes incision, using the available ossicular remains plus the addition of polyethylene tubing to insure passage of sound from the tympanic membrane to the perilymph of the vestibule.

Perforations of the tympanic membrane, regardless of size, are closed with vein grafts which our preliminary use indicates are superior to skin but more difficult to procure in adequate size. These grafts act as a tissue splint on the undersurface of perforations and seem to encourage an epithelial outgrowth from the edges of the perforation.

With total absence of a tympanic membrane, I have had success using a combination of skin and vein graft for replacement. The skin is best obtained from the lower meatal wall and rotated on a pedicle to cover the dehiscence with a vein underneath supported by Gelfoam to blend with the middle ear mucosa.

Regardless of the method, the ultimate goal after elimination of disease is restoration of hearing and preservation of anatomic structures so the ear is returned to a near normal state.

Doctor Pou has demonstrated that this is attainable in a series of his cases, and I want to compliment him for directing our thinking in the future more along these lines.



CASE REPORT

Congenital Patent Urachus

R. T. McCAULEY, M.D., *Danville, Va.,* and F. R. LICHTENHELD, M.D., *Wiesbaden, Germany*

PATENT URACHUS results from failure of the allantoic duct to close and is frequently secondary to an obstruction at the vesical outlet or distal to this. The cloaca is an entodermal reservoir which is present in vertebrates below the level of the Placentalia. Into this common receptacle the waste products of the fecal and urinary tracts are emptied as well as the products of the reproductive tract. In the higher animals the cloaca is divided into a dorsal rectum, a ventral urinary bladder, and a urogenital sinus by growth of the rectovesical pouch and septum. By the sixth week in the human embryo the cloaca has divided into two portions; a dorsal segment to form the rectum, and a ventral segment which becomes the urinary bladder and posterior urethra. The upper portion of the ventral division enlarges to form the bladder and receives the allantois. The lower portion of the ventral division becomes the entire urethra in the female and the prostatic urethra in the male. The urachus passes from the uppermost portion of the bladder to the umbilicus and is formed by a condensation and fibrosis of the allantois. The structure is situated between the peritoneum and the transversalis fascia in the midline and is accompanied on either side by the obliterated hypogastric arteries. Rarely the urachus fails to close, thus giving rise to a fistulous tract extending from the apex of the bladder and emptying at the umbilicus.

The original observation and description of congenital patent urachus is said to have been made by Cabrolius[1] in 1550. Urachal cysts and acquired urinary fistulas at the umbilicus should neither be confused with nor classified as patent urachus.

Congenital patent urachus is a rare condition. Nix, Menville, Albert, and Wendt[2] reported that the diagnosis was made only three times in over a million admissions to Charity Hospital in New Orleans between the years 1936 to 1956. The diagnosis has been made twice at the USAF Hospital in Wiesbaden, Germany, since 1945. During this period over 108,000 patients were admitted. It occurs two to three times more frequently in males than in females. Both patients seen at this hospital have been male.

The diagnosis of congenital patent urachus is usually very easily made by observing the leakage of urine from the umbilicus following the separation of the cord. This urinary leakage may vary from a few drops to a fully flowing stream. It is likely to be most apparent when the child is straining or voiding. The umbilicus may be relatively normal in appearance or may be involved in a tumor-like process consisting of the stump of the cord which is being nourished by the urachal arteries. At other times there may be an associated umbilical hernia, neoplasm or calculi. Leakage of urine may cause excoriation or irritation of the periumbilical skin and if infection is present, these skin changes may be more marked.

The presence of an unusually large cord or other anomaly at birth is commonly associated with a patent urachus. Reflux of urine through the urachus into the cord can cause cystic dilatation of the cord prior to separation.

The presence of a patent urachus can be proven in one of several ways. Proof by analysis that the draining fluid is urine is probably one of the simpler methods. The introduction of a dye such as methylene-blue or indigo carmine with its subsequent passage through the urethra is another simple diagnostic test. Discoloration of the umbilical fluid following the injection of indigo carmine intravenously, introduction of a small

FIG. 1

Cystogram (lateral view). Elongated bladder with partially obliterated sinus tract toward umbilicus.

catheter through the umbilicus into the bladder, and injection of a radiopaque medium either through the patent urachus or through the urethra are often diagnostic. Cystoscopic examination is helpful in determining any obstruction in the lower urinary tract, and accurate in observation of the size of the patent urachus. Excretory pyelograms determine the presence of any upper urinary tract anomalies.

Many forms of treatment have been attempted in the past, including cauterization, ligation, excision, and combinations of these. The best form of treatment, however, is extraperitoneal excision. This can be readily done through a midline incision with a small probe in the fistulous tract or by injecting it with methylene-blue. A patent urachus should be excised shortly after the diagnosis is made since the incidence of attendant infection is quite high. If there is any obstruction of the lower urinary tract, it should be corrected previous to or at the time the patent urachus is excised.

Case Reports

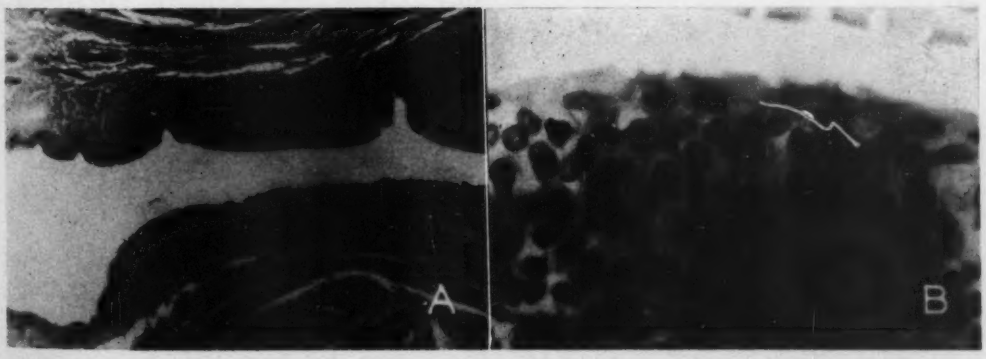
Case 1. C. L., a 2-month old negro male, was admitted to the United States Air Force Hospital, Wiesbaden, Germany, on September 25, 1957. The complaint was the intermittent passage of clear fluid several times a day from the umbilicus since shortly after birth.

Physical examination revealed a small umbilical hernia with a drop of clear fluid appearing on pressure on the lower abdomen. Urinalysis was normal but a mild anemia existed. Excretory urography disclosed normal upper urinary tracts and the x-ray film of the lungs was considered to be within limits of normal. Cystourethrograms illustrated a high bladder with a urachal fistula. The vesicourethral junction was interpreted as being within normal limits. (The cystourethrograms were taken by injection of contrast media through the urethra.) Surgical correction of the urachal fistula was deferred because of an upper respiratory infection and diarrhea.

Upon a second admission on February 4, 1958, the umbilicus was found to be dry and pressure on the lower abdomen did not produce any urinary seepage. Cystogram revealed an elongated bladder with a small partially obliterated sinus tract extending from the apex of the bladder toward the umbilicus (Fig. 1). No contrast medium extravasated on the abdomen. Observation of urination indicated a forceful good stream. It was thought that the urachal fistula had closed spontaneously, the bladder emptying itself completely with each urination and that the vestigeal sinus tract would spontaneously close with time. The patient was discharged from the hospital.

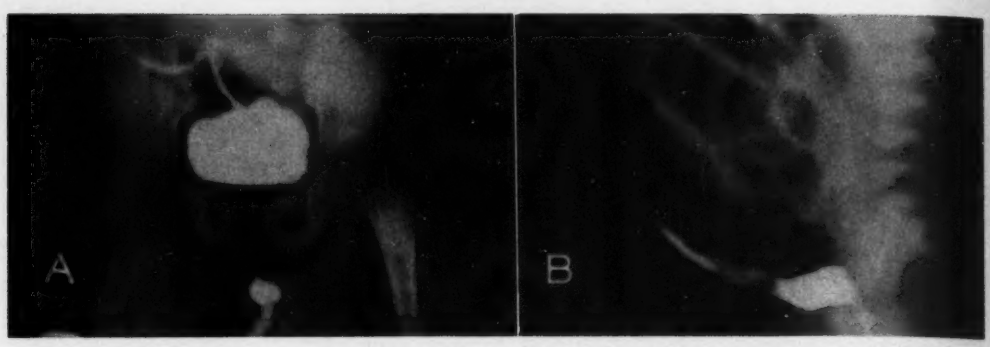
However, 2 months later on April 14, the child was brought back to the hospital draining urine from the umbilicus. Extraperitoneal excision of the patent urachus, dilatation of the vesical neck and repair of the umbilical hernia were performed. The postoperative course was uneventful and the patient was discharged 9 days following the last admission. Further

FIG. 2



(A) Low power magnification of section through patent urachus. (B) High power magnification of section through patent urachus.

FIG. 3



(A) Cystogram (AP view). Catheter is inserted through the umbilicus into the bladder. (B) Cystogram (lateral view) demonstrating large patent urachus.

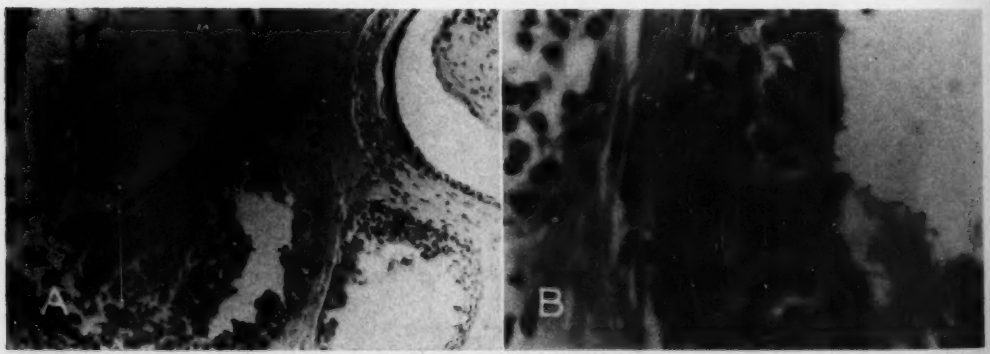
observation indicated that no further difficulty was experienced. The fistulous tract was 4 cm. long, 3 mm. in diameter at the skin surface and 8 mm. at the bladder area. The histologic interpretation of the tube-like structure showed normal transitional epithelium similar to that found in the urinary bladder (Fig. 2,A and B).

Case 2. G. K., a 3 week old male infant delivered by cesarean section in Karachi, Pakistan, weighed 6 pounds at birth. At age 2 weeks the parents indicated that oozing of clear fluid from the area of the umbilicus had been noted. Confirmation that the oozing material was urine was obtained by injecting 1 cc. of indigo carmine solution intravenously and observing the dye appear at the umbilicus in 3 minutes. He was admitted to the USAF Hospital in Wiesbaden, Germany, on September 11, 1959. The area of the umbilicus disclosed small amounts of clear drainage oozing from what was interpreted as a patent urachus.

The laboratory studies revealed that the urine contained white blood cells too numerous to count, a leukocytosis of 21,950 with 28% neutrophils, 10% bands, 86% lymphocytes and 4% eosinophils. *Bacillus proteus* was cultured from the urine. A cystourethrogram obtained as described in the above case demonstrated a normal vesicourethral junction as well as patency of the urachus. A catheter was also passed into the bladder through the patent urachus (Fig. 3, A and B).

Six days following admission extraperitoneal excision of the patent urachus was performed. The postoperative course was characterized by the development of atelectasis and pneumonitis of the left upper lung field. He also developed a rather severe paralytic ileus. With rather vigorous treatment he gradually improved and was able to resume oral feedings. On the 4th day following operation the urethral catheter inadvertently came out and urine drained through the suprapubic wound. However, the drainage subsided during the subsequent several days and he was able to void through the urethra satisfactorily. Epididymitis developed on the right side, but this improved by the time of his discharge from the hospital on November 1, 1959. The ultimate response to surgery was gratifying, since the urine had lost its infection and the patient was gaining in weight. The pathologic examination confirmed the diagnosis of congenital patent urachus. There were six lumens lined by transitional epithelium in edematous and subacutely inflamed smooth muscle and fibrous tissue (Fig. 4,A and B).

FIG. 4



(A) Low power magnification of section through patent urachus. (B) High power magnification of section through patent urachus.

Discussion

Patent urachus usually does not present a diagnostic problem, if leakage from the umbilicus is present. After the diagnosis has been established by the procedures described, obstruction of the bladder neck should be ruled out if a good postoperative result is to be obtained. The hope for permanent spontaneous closure of the patent urachus is quite vain, as was demonstrated in case 1. Surgical dissection of the patent urachus should be carried out, since recurrent infections of the urinary tract are quite common if operation is not done. Even long-term medication may not prevent the original infection from developing into a chronic pyelonephritis.

Summary

Patent urachus is a rare congenital anomaly, being observed only twice during the past 14 years at the USAF Hospital, Wiesbaden, Germany, during which time there were over 108,000 admissions to the hospital. The development, diagnosis, and treatment of congenital patent urachus have been discussed. Two previously unreported cases have been presented.

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Fifty-fourth Annual Meeting

Southern Medical Association

St. Louis, Mo., Oct. 31-Nov. 3, 1960

Osteogenesis Imperfecta Treated by Fixation with Intramedullary Rod

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OSTEOGENESIS IMPERFECTA is a generalized disease of the mesenchymal tissue, one factor of which results in weakening of the skeletal system. The disease is one of osteoblastic inability to produce mature osteoid. No known form of treatment to correct the basic lesion is known. Fractures produced by osteogenesis heal normally, but produce the same weakened bone that was present prior to injury. Often multiple fractures and muscle pull result in deformities of such severity that ambulation is impossible.

The differentiation of the disease into, (1) congenital and (2) tarda groups is accepted by most authorities in the field, with the congenital type the more severe with multiple intrauterine fractures often resulting in neonatal death. The tarda group varies from multiple fractures in childhood, due to insignificant trauma, to a group of patients in which there seems to be a greater tendency to fracture than in other children in the same age group. In the latter group blue sclera, generalized thinning of the cortices, and a deficiency of cancellous bone may be present to varying degrees. Many of these patients can be handled by routine immobilization in plaster and no problem in management arises.

However, there is another group of patients in which the disease is so severe that routine immobilization is inadequate. Fractures in this group render them completely bedridden in a spica. There have been 5 patients at the Shriners Hospital, and one at the Confederate Memorial Medical Center in Shreveport, who have been treated by Sofield method of fragmentation, use of a rod and realignment. An intramedullary rod is reserved for the patients with severe involvement or severe deformity. It is with this group of patients that this paper concerns itself.

One patient seen since this treatment was started in 1947, because the disease and the deformity were so severe, was refused the use of an intramedullary rod. Several patients during the same period have been handled by plaster immobilization in the generally accepted manner.

The findings and recommendations in this paper are based on experience in 6 patients ranging from 2 to 8 years of age. All had had multiple fractures resulting in severe deformities. Bilateral femoral osteotomies and insertion of rods have been done in the 6 patients. Because of overgrowth or subsequent fractures or angulations there have been 8 secondary operations. Bilateral tibial osteotomies and insertion of rods have been done in 4 of the patients, and there have been 2 instances in which subsequent rods needed to be used. Three of the patients are walking with braces and crutches. One patient is walking with braces only, one patient is walking with a walker, one patient does not walk. Figures 1 and 2 are illustrative of pre- and postoperative findings and results.

In summary, 6 cases have been presented to illustrate the use of intramedullary rods in the lower extremities for osteogenesis imperfecta. It is appreciated that there are many disadvantages to this treatment, mainly, that it is a formidable operation attended by all its inherent risks, and that multiple procedures will be necessary as the patient grows. It was found true that many revisions are necessary, once this type of treatment is begun at an early age. However, the younger the patient the more likely he is to develop severe involvement. Fractures and angulation do occur after rods have been used, but they are easier to treat with much less resultant deformity. In many patients braces are necessary as an adjunct to ambulation.

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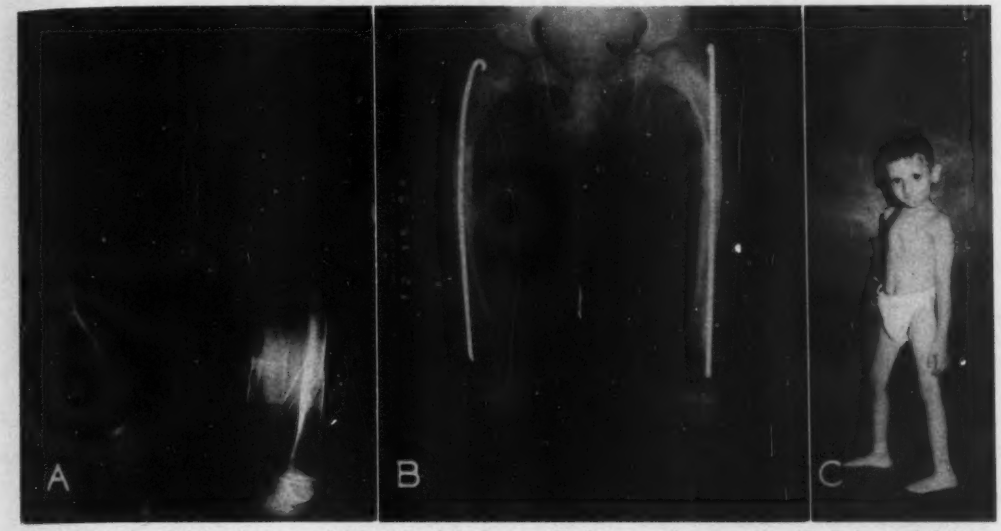
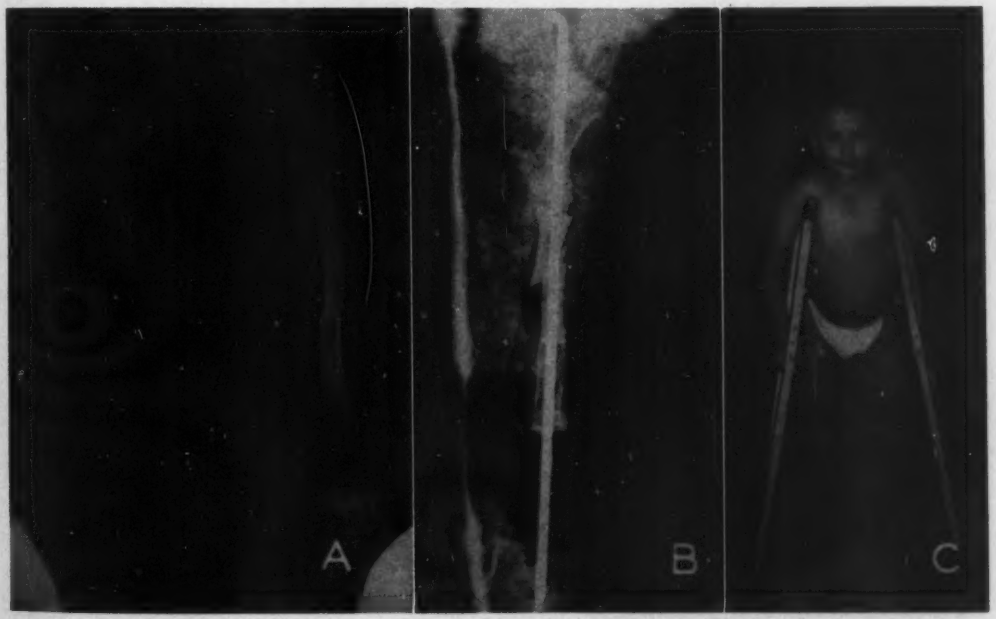
FIG. 1



(Case 1) (A) On admission, fractures and deformities of the femurs. (B) One year after operation. (C) Patient ambulating unassisted.

Although the treatment of osteogenesis imperfecta by the method of fragmentation, the use of rods and realignment in the manner of Sofield does not attack the basic disease, it is the best-known aid for the prevention of severe deformities and providing for walking and more nearly normal activity. It is expected that further experience with this form of treatment will aid in the proper selection of patients and optimal time for operation.

FIG. 2



(Case 4) (A) On admission, fractures and deformities of the femurs. (B) Immediately postoperative in plaster, showing fragmentation, rod and realignment. (C) Walking in double upright braces and crutches; he had never walked until treated.

Parenteral Dexamethasone Phosphate in Allergy

WILLIAM C. GRATER, M.D., *Dallas, Tex.*

The author is favorably impressed by the parenteral use of this steroid in acute manifestations of allergy.

Introduction

INJECTABLE CORTICOSTEROID THERAPY has proven useful in a variety of allergic disorders. Some of the more common situations where these preparations may be used effectively include:

(1) Life-threatening allergic emergencies, such as status asthmaticus, laryngeal edema and anaphylactic reactions. (In the latter situations, of course, parenteral steroids should not be used to the exclusion of epinephrine.)

(2) More benign acute allergic disorders, where rapid relief of symptoms is desired, such as acute drug reactions.

(3) Patients who cannot take oral medication because of vomiting, anesthesia, or heavy sedation. This also includes the treatment of very young infants.

(4) Adrenal Insufficiency. Although classical Addison's disease is seldom seen by the allergist, the widespread use of adrenal steroids can lead to a number of cases of relative insufficiency of iatrogenic origin.

The pharmaceutical preparations available for this purpose have been improving steadily. Intravenous hydrocortisone alcohol was the first commercially available preparation which proved useful.[1] However, it was relatively dilute, was dissolved in 50% alcohol and had the electrolyte problems common to all hydrocortisone therapy. Water soluble prednisolone was definitely a superior preparation. Now the phosphate salt of dexamethasone is available in aqueous solution in 4 mg. per cc. multiple dosage vials.* It may be given by any injectable route: subcutaneous, intramuscular and intravenous. The phosphate esters of the various steroids have been shown to be more rapidly absorbed than the free alcohol or other available esters.[2] This permits the large loading dosage necessary for the prompt suppression of allergic reactions. It is also necessary when stress complicates hypoadrenalism.

This report deals with clinical experiences using injectable dexamethasone phosphate, by the subcutaneous and intramuscular routes.

Methods

A sterile solution of dexamethasone phosphate in concentration of 4 mg. per cc. was used to treat 40 patients who had various acute allergic disorders (Table 1). In 3 of these patients the acute allergic exacerbations were thought to be associated with hypoadrenalism incident to the sudden cessation of oral steroid therapy. The usual initial dose was 1 cc. (4 mg.), although as much as 12 mg. was given on occasion. The subjective response was recorded and the objective response measured by means of timed vital capacities in asthmatic subjects, and Coreco Kodachromes in patients with dermatologic disorders. The patients were maintained on

TABLE 1

PARENTERAL DEXAMETHASONE IN ALLERGIC DISORDERS

Disorder	*Satisfactory Results*	*Unsatisfactory Results*
Allergic rhinitis (acute pollenosis)	4	0
Asthma	24	1
Atopic dermatitis	1	1
Contact dermatitis	4	0
Drug allergy	1	0
Urticaria	1	0
Acute hypoadrenalism (after steroid therapy)	3	0
Totals	38	2

*Supplied as injection Decadron phosphate by Dr. Nicholas Capeci of Merck Sharp & Dohme Research Laboratories.

parenteral dexamethasone, but shifted to oral therapy as soon as possible.

Results

As seen in table 1, the parenteral use of dexamethasone phosphate produced a satisfactory amelioration of symptoms in 38 of 40 patients. It was difficult to be sure of the onset of action, since all patients were placed at rest in a pollen-free room which may have contributed to their improvement. Moreover, the objective measures of improvement are not always extremely sensitive. Nonetheless, we had the impression that the onset of action was rather rapid. Subjective improvement was often noticed within one hour and objective improvement recorded within four hours.

No difference was noted in the rapidity of onset of improvement following intravenous injections when compared to subcutaneous or intramuscular administration. It should be noted, however, that none of the patients were in frank shock at the time of treatment. The duration of activity of the compound was approximately 12 hours. This permitted a "every 12 hour" dosage schedule for as long as parenteral therapy was required. In severe situations, as a precaution, treatment was sometimes given more frequently.

Nervousness and insomnia were noted in 2 patients. These symptoms subsided when the dose was reduced. No other side effects were recorded, and there was no instance of local irritation from the injections by any route.

Case Reports

Case 1. G. J., a 69 year old man with widespread contact dermatitis, was weaned from corticosteroids for patch testing. He had a severe exacerbation with burning, itching and widespread dermatitis. The only medication consisted of dexamethasone 4 mg. I.M. followed by 8 mg. 1½ hours and 8 mg. 6 hours later. For the next week he was given 8 mg. intramuscularly daily, and then much lower dosages of oral dexamethasone. Results were dramatic with subjective relief of pruritis in 1 hour. Objective results were noted in 6 hours (Fig. 1).

Case 2. J. A. was a 3½ year old, 37 pound boy with status asthmaticus, which was resistant to aminophyllin and epinephrine. He was afebrile, was vomiting, and presented a shock-like picture. In the hospital he was treated with oxygen, I.V. fluids and penicillin without results for 6 hours. Then he was given 4 mg. dexamethasone subcutaneously. Marked clinical improvement was shown in 3 hours. Dexamethasone was continued subcutaneously every 6 hours for 48 hours when he was transferred to oral dexamethasone from which he was then weaned. He had never received systemic steroids previously. His life may have been saved by injectable corticosteroid therapy.

Discussion

Because of the well-known risks attendant upon *chronic* steroid therapy, one may occasionally forget the important and sometimes

FIG. 1

(Case 1) Acute disseminated neurodermatitis. (A) Before therapy. (B) Dexamethasone 2 cc. I.M. was given every 12 hours. Photograph 24 hours later. (C) Four days later.

life-saving role these compounds play in the treatment of *acute* disorders. In such situations, moreover, they can and should be given in relatively large doses since the occurrence of undesirable steroid effects is negligible in very short-term therapy. In view of the relative safety of short-term therapy, the use of injectable steroids need not only be limited to emergency situations, but may also be used to relieve acute discomfort. Following the injection of dexamethasone phosphate (even by the subcutaneous or intramuscular routes), the patient may experience relief more quickly than when treatment is initiated with oral medication.

In the United States, enormous numbers of people have been given steroids systemically for one reason or another. The possibility of hypoadrenalism in times of stress must always be considered. Three patients in the present study had a relative state of hypoadrenalism, and a good response to injectable dexamethasone phosphate was observed. It was interesting that the patients often had subjective relief before objective relief could be noted by the physician. It is, of course, possible that the methods for measuring objective relief are relatively crude.

Summary

1. Injectable dexamethasone phosphate proved a highly satisfactory preparation in 38 of 40 patients suffering from acute allergic disorders. Three of these were complicated by hypoadrenalism after steroid therapy. The side effects were significant.

2. This preparation is useful in patients requiring corticosteroid therapy who are unable to take oral medication, or in whom rapid onset of steroid activity is desired.

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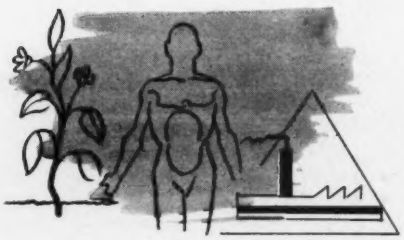

CASE REPORT

Acute Glaucoma Following Extraocular Surgery Despite Definitive Ocular Preventive Measures*

SHERMAN B. FORBES, M.D., *Tampa, Fla.*

Here is a warning. In the patient having glaucoma and needing an operation of a general nature, medication preparatory to general anesthesia, as well as certain postoperative factors, may present a hazard in terms of acute glaucoma. Patients under these circumstances deserve the careful observation of an ophthalmologist.

GARTNER AND BILLET[1] recently directed attention to the occurrence of acute glaucoma as a complication of major extraocular surgical procedures. They described four cases and cited a number of cases reported in the literature in which this complication occurred. There was no mention of the immediate preoperative condition of the eyes in their cases, but the patients were unaware of any eye trouble prior to operation.

Schwartz, de Roetth and Papper[2] reported the effects on intraocular pressure produced by the intramuscular injection of therapeutic doses of atropine sulfate and scopolamine hydrobromide in 14 patients with chronic simple (open angle) and acute (angle closure) glaucoma. Both drugs produced a slight rise in intraocular pressure in but 5 of 28 eyes, an increase regarded as insignificant, while in 23 eyes there was no change or a decrease in the tension. From the results in their series these authors concluded that the safety of a single dose of parenterally given belladonna drugs for preoperative medication in glaucomatous patients was clearly established. They could find only one case in the literature in which preanesthetic injection of atropine in reasonable dosage may have contributed to the erroneous belief that parenterally given atropine and scopolamine are contraindicated for preanesthetic medication in patients with glaucoma. They recommended the topical instillation of pilocarpine hydrochloride (1 to 2%) or physostigmine salicylate (0.25 to 1%) into the conjunctival sac in the preoperative and postoperative periods to produce miosis to counteract the potential mydriatic effect of the belladonna drugs and the anesthetic agents. They postulated that in the safe management of patients with glaucoma for extraocular operation, morphine sulfate and the barbiturates may be used as premedication and during general anesthesia.

It may be then that the case here presented is the second one to be reported in which all the criteria laid down were carried out in using scopolamine for preoperative medication in a glaucomatous patient with a resultant acute episodic phase of the disease in both eyes. The patient had been under my supervision over a number of years and, prior to undergoing a pelvic operation, was known to have a chronic wide angle glaucoma associated with severe migraine and a psychopathic pattern. In spite of active preventive measures locally, a severe acute attack of glaucoma occurred in the postoperative period.

Report of Case

A white woman, aged 42 years, first consulted me in 1949 for a routine refraction. There was a history of migraine and psychopathic disturbance, both of which had required active treatment.

On ocular examination, there was a high hyperopic

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refractive error with vision easily correctible to 20/20 in each eye. The findings otherwise were negative, including a normal tonometric reading of 20.4 mm. Hg. (Schiøtz) in each eye. The anterior chambers were fairly deep. When the patient was examined again the following year, there were no significant symptoms or clinical findings. The next examination was made in 1954, at which time she complained of a migraine type of headache. The intraocular pressure was 16.9 mm. Hg. in the right eye and 20.1 mm. Hg. in the left eye.

In January 1956, the patient consulted me again, complaining of severe migraine attacks and of seeing rainbows around lights at the time of these episodes. The objective examination revealed little. Studies of the visual fields gave normal results for form and color in both eyes, and the blind spots were of normal size. The pupils were dilated with cyelopentolate HCl (Cyclogyl) with a resultant intraocular pressure of 62.9 mm. Hg. in the right eye and 48.3 mm. Hg. in the left eye and much ocular discomfort. The pupils were contracted with eserine, and the patient left the office with the pressure normal. A few days later, gonioscopy showed a plateau type of iris with a fairly open angle and deep chamber, a considerable number of peripheral anterior synechiae and some pigment. The facility of aqueous outflow was 0.15 cc. in the right eye and 0.10 cc. in the left eye. A test in the dark room during one of the migrainous episodes showed the tension elevated from 20.1 mm. Hg. in both eyes to 62.9 mm. Hg. in the right eye and 55.0 mm. Hg. in the left eye. The water drinking test gave positive results in moderate degree in both eyes. Pilocarpine HCl (2%) to be used on retiring, and massage several times daily were prescribed. Two months later a test in the darkroom gave equivocal results.

One month afterward, in June 1956, the patient was told that a hysterectomy was necessary. Advice was sought in reference to the ocular pattern during this procedure. The ocular tension was 16.9 mm. Hg. in both eyes. She had used neither ocular medication nor massage for one month. On the day before the operation, pilocarpine HCl (2%) was used in both eyes 4 times. An instillation was made just prior to the induction of the anesthesia. Routine preoperative medication was given, pentobarbital sodium (Nembutal) (200 mg.) the night before the operation, and on the morning of the operation Nembutal (100 mg.) at 7 a.m., and meperidine HCl (Demerol) (100 mg.) and scopolamine (0.02 gr.) at 8 a.m. Administration of the anesthetic was started at 9 a.m. The intraocular pressure, taken when the patient was under the surgical stage of anesthesia, was 14.2 mm. Hg. in the right eye and 16.9 mm. Hg. in the left eye. The pupils were of normal size for this patient. Pilocarpine HCl (2%) was used in both eyes at the time of this last tonometric evaluation under the anesthetic.

On the first postoperative day the patient complained bitterly of pain particularly in the left eye, stating that the abdominal discomfort did not compare with the ocular distress. There was an acute episode of glaucoma in both eyes with corneal edema, a half dilated pupil in the right eye and a fully dilated and fixed pupil in the left eye. The intraocular pressure was 48.3 mm. Hg. in the right eye and 62.9 mm. Hg. in the left eye. Isoflurophate (Floropryl) (0.1%) was used in both eyes the evening after the operation and again the next morning. Acetazolamide (Diamox) (250 mg.) was given once intramuscularly and three times by mouth on the first postoperative day. On the second postoperative day, local medication consisted of pilocarpine HCl (2%) 4 times and Diamox (250 mg.) 3 times by mouth. As the glaucoma was obviously controlled, the pilocarpine HCl and the Diamox were continued during the hospital stay.

For 3 months following the operation there were variable findings, usually including a normal intraocular pressure with an equivocal reaction to dark room and also water liability tests. A positive blockage of the angle with pigment deposition in the angle, increase of the iris plateau pattern, and a considerable number of peripheral anterior synechiae developed. A basal peripheral iridectomy was therefore performed, with a resultant corrected distance vision of 20/20 plus in each eye and repeated tonometric readings for months after the operation ranging around 15 mm. Hg. in each eye without therapy of any kind. The peripheral visual fields were normal for form and color, and the blind spots were practically normal in size. There was an associated improvement in the migrainous and psychopathic symptoms, and perhaps the improvement in the emotional stability was a factor in the improved condition of the eyes.

The last observation was made 2 years and approximately 3 months after the ocular surgical procedure, at which time the corrected distance vision was 20/20 plus in the right eye and 20/20 in the left eye. The intraocular pressure was 16.9 mm. Hg. in the right eye and 23.4 mm. Hg. in the left eye. The facility of aqueous outflow was 0.15 cc. in the right eye and 0.125 cc. in the left eye. There was a definite unusual atrophy of the iris stroma above to the temporal side in the right eye and in multiple areas to a greater degree in the left eye. Gonioscopy showed the entire angle to be clouded and somewhat blocked except in the region of the iridectomy. The basal iridectomy was obviously functioning well with no pigment blocking the bypassing of the aqueous flow. At this time the reaction to the water drinking and Euphthalmine provocative tests was positive to a much lesser degree than in 1956.

Comment

In this case, a highly malignant episode of glaucoma was apparently precipitated by the preoperative preparation associated with a general surgical procedure together with the emotional strain of undergoing a hysterectomy in a patient with a mild form of chronic wide angle glaucoma of a fluctuating type. This case may represent an episodic disease to some extent associated with either a migrainous or a psychopathic disturbance, or both. It was impossible to overlook the immediate postoperative episode of glaucoma because of the intense pain. Although pilocarpine HCl (2%) was used several times before the operation and in the operating room, this treatment did not prevent an acute attack.

It is of course difficult to explain the acute episode in this case of wide angle glaucoma, but in the light of a recent article by Flocks[3] it becomes more understandable. He proposed a new theory on the pathogenesis of primary wide angle glaucoma, namely, degenerative changes in the trabecular meshwork associated with diurnal variations of intraocular pressure. Summarizing this theory, he stated that primary open angle glaucoma is caused by the presence of vulnerable trabeculae or a pressure-regulating mechanism which is damaged by levels of intraocular pressure which ordinarily do not damage the trabeculae or optic nerve. This injury interferes with aqueous drainage and promotes an increase in intraocular pressure which further damages the trabecular meshwork. He concluded that in his series of cases "the location and character of the degenerative changes indicate that they are responsible for the decreased facility of aqueous outflow characteristic of primary open-angle glaucoma."

In the case reported here these changes probably were present, and also there was a gradual development of peripheral anterior synechiae in the angle. In early 1956 there was a very positive reaction to a mydriatic provocative test accidentally observed, and a few days later the result of a dark room test was likewise highly positive—the two most valuable tests of angle crowding. In addition, a positive response to a water drinking test and a decreased facility of aqueous outflow were observed, all showing there was in this case of wide angle glaucoma a secondary crowding of the angle and blockage with a considerable degree of pathologic change at the angle including trabecular sclerosis. Superimposed clinically was a highly psychotic and migrainous pattern of a cyclic nature. In discussing this case, two of my colleagues mentioned essential atrophy of the iris with the degree of atrophy of the iris which eventually occurred. Engorgement of the angular vessels of a cyclic nature should be considered. It would appear that the pattern of glaucoma in this case may be largely explained on a neurovascular basis, particularly as there was always a low facility of aqueous outflow with probably cyclic variations in the rate of aqueous production.

The absolute control of the glaucoma with the peripheral iridectomies may suggest there was some degree of pupillary block as well as angular obstruction. A pupillary block certainly could be considered with particularly the iris changes which occurred.

Were I to encounter another case of this type, I should be inclined to use more active topical antiglaucomatous therapy associated possibly with the administration of Diamox or some other carbonic anhydrase inhibitor throughout the preoperative and postoperative period. I should also try to prevent the preoperative use of atropine or scopolamine and certainly should suggest that succinylcholine not be used.

My impression is that it is the duty of ophthalmologists to take a more active part in directing the much needed preventive medication when their patients with glaucoma undergo nonocular operations in an effort to avoid an acute attack of the disease. Otherwise, in some cases the control of the glaucoma may not be as successful as in the case presented.

Summary

A case is reported in which a patient with chronic wide angle glaucoma experienced an acute episode of the disease, despite preoperative preventive measures, on the first postoperative day after being subjected to a hysterectomy. It is suggested that the pattern of glaucoma in this case may be largely explained on a neurovascular basis.

It is concluded that it would be well for the ophthalmologist to assume a more active role in supervising preventive measures to guard the patient with glaucoma who undergoes extraocular surgical procedures from an acute attack possibly precipitated by medication incident to the operation.

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Discussion (Abstract)

Dr. Albert C. Esposito, Huntington, W. Va. Dr. Forbes is to be congratulated in bringing this unusual case to our attention. His interest in glaucoma is not new for, as you may recall, he did some of the pioneering work on cyclodiathermy and his description of this surgical technic has been incorporated verbatim in one of our newer textbooks on ocular surgery.

The case presented is well documented and is the only one of a wide angle glaucoma to be so recorded.

The mechanism of its formation fits into Flocks' theory of trabecular degeneration but cannot accurately be known without serial sections of the globe in question. Gartner and Billet recorded 4 cases of narrow angle glaucoma following general surgery, but the pattern in all these 5 cases remains similar. Knowing the predisposing causes for an attack of acute glaucoma in both narrow and wide angle glaucoma, it becomes apparent that the conditions for its appearance are definitely present both prior to and following general surgery. It is possible that transient acute attacks of glaucoma do occur but are missed by the general surgeon.

The factors conducive to the production of acute glaucoma at the time of general surgery should be considered. Atropine or scopolamine usually are given routinely before general anesthesia to reduce the secretions in the airways, and it is possible that even the small doses usually employed may cause sufficient pupillary dilatation to produce angle closure glaucoma in a predisposed individual.

Further, all general anesthesia, and especially ether, produces contraction of the pupils in the early stages of anesthesia while in deeper stages pupil dilation takes place.

We know that succinylcholine, which is similar to curare, produces muscle relaxation and is used in general surgery. It produces as a side effect, a rise in intraocular pressure which can also produce the acute attack. (Hofman et al., 1952.)

Another factor present is the patient's excitement in being hospitalized and awaiting operation. This emotional strain alone may be a precipitating factor in producing the acute attack of glaucoma. Our Chairman-Elect, Dr. Haik, in his paper before the American Academy of Ophthalmology and Otolaryngology this year, documented this fact quite clearly.

Following general surgery, the patient usually remains in semidarkness which may again act as a darkroom test does, in dilating the pupil and blocking the chamber angle as the mechanism in the predisposed narrow angle.

These various factors then may all or individually produce the picture of acute glaucoma and, because of the expected nausea and vomiting after operation, the picture and the symptoms may not be recognized, and the patient's discomfort blamed on the anesthesia and the operation.

Gartner and Billet, and others, recommend that, prophylactically, pilocarpine be instilled both prior to and after operation; however, as noted in Dr. Forbes' paper, he followed this technic in his case of open angle glaucoma but the attack of acute glaucoma was not prevented.

All 5 cases reported responded to medical treatment initially, with 3 cases, including this one, going on to operation before complete arrest of the condition. This paper should certainly make us more aware of this unusual and rare complication to general surgery.

I thoroughly enjoyed Dr. Forbes' most excellent paper. I thank you.

The Aging Eye and Entoptic Phenomena*

A. E. MEISENBACH, SR., *St. Louis, Mo.*, and
A. E. MEISENBACH, JR., *Dallas, Tex.*

With an accumulation over the years of drawings of these phenomena as described by patients, the author provided a visual description for the listener of what the patient sees.

As OPHTHALMOLOGISTS we have at times had patients describe vaguely visual phenomena such as cobwebs, specks, flashing lights, colored halos, floating bodies, partial loss of the field of vision, etc. Very few are able to give any accurate description of their symptoms. The purpose of this paper is to document by actual drawings the visual phenomena, entoptic and otherwise, which have occurred to one of us (A.E.M., Sr.) over a period of many years and to illustrate their development over a period of time.

Migraine

The reports on migraine go back into medical antiquity and the reports of Sir Hubert Airy,[1] Liveing,[2] Jolly,[3] Jelliffe,[4] Duke-Elder,[5] and most recently Miles,[6] as well as many others, have well documented this visual phenomenon. It is not our purpose to discuss the various theories advanced for their explanation. Most attacks last about 20 to 25 minutes and consist of flickering colored and white zigzag lines, followed by a negative scotoma best observed on printed matter.**

The central origin of scintillating scotomas is now generally conceded. However, it is in no way related to retinal arterial spasm except that both are due to an angioneurosis. It is also thought that heredity is a factor in the development and continuation of hemicrania. Migraine may be defined as a symptom complex (paroxysmal in nature) characterized by hemicrania and symptomatic evidence of cortical involvement. All migraine is headache, but not all headache is migraine.[7]

The appearance of the expanding scotoma and the so-called "fortification phenomena" is illustrated on a white background. The expanding form of a scintillating scotoma can take many unusual forms, and may have a varying bluish-brown in the inhibited zone with marked variation in the zigzag line.

The tendency to break down migraine into various divisions such as: (1) hemicrania, (2) facioplegic, (3) ophthalmic, (4) psychic, (5) ophthalmoplegic, and (6) abdominal, hardly seems justified. For discipline purposes such classification may be desirable but the fundamental etiologic factor underlies manifestations in all types. Migraine commonly stops at middle age. Scintillating scotoma does not.

I have personally experienced on several occasions a most unusual phenomenon consisting first of everything having a reddish glow, like looking through a thin smear of blood. This was constant, restricted to only the left eye and gradually deepened in color without loss of visual acuity over a period of some 30 to 40 minutes. Subsequently there occurred a marked dazzling effect, so that looking out into the bright sunlight all objects had a brilliant canary yellow outline with some distortion of central vision. When looking at evergreens or cedar trees they had a dark brown dead-looking appearance, but other colors were not affected. This lasted for nearly one hour following which the phenomena gradually disappeared with no sequelae. This has occurred three times over a wide interval of time.

Airy,[1] Latham,[8] Jolly,[3] Wheatstone,[9] Lashley,[10] and Purkinje have all described the expanding figures of scotoma scintillans as is shown here on slides. These show the phenomenon in its spectral colors to a pure white

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**The presentation was illustrated by 72 Kodachromes which unfortunately cannot be reproduced for the printed page.

only, as described by Lashley. His had a peculiar expanding kidney bean shaped form. Purkinje reported the occurrence of flickering circles and bands after taking large doses of digitalis for four days.

Slides illustrate: (a) the chromatic expanding type with the light luminous against a dark background and dark against a light background; (b) a chromatic large area showing development state of inhibited zone and later stage in which the inhibited zone is clearing up; (c) partial chromatic expansion but chiefly achromatic; and (d) how widely these scotoma scintillans can vary from the usual spectral colors and "fortification phenomena" so frequently described in the literature.

Gowers, in his discussion of migraine, describes the rainbow phenomenon and a zigzag pericentral spectrum around his dinner plate, like a halo which would flash up and pass out of his field of vision overhead as he would look up. In addition he described progressive, radial, and medial types with vision retained in clear areas and inhibited in shaded areas. There may be four phases of a pericentral spectrum with a uniform diffuse to a jagged chromatic spectrum and luminous central area with a ring of vision around it and further surrounded by a dark area.

Another unusual form of this scotoma is one in which only white appears. The figure retains its complete outline with no spectrum. Some remarkable figures, with cross hatched interiors which are all white and retain their complete outline, may occur. These are very unusual, difficult to classify, and resemble the pericentral type described by Gowers. Unusual forms of a partial expanding chromatic scotoma and a reverse of the interior to exterior may occur.

The entoptic visualization of the optic nerve and retinal fibers are an after-image effect. Peculiar brownish tinged corpuscular bodies may offer a shower of particles, moving with great rapidity in the visual field.

Early Lenticular Changes

In the slides showing cataractous halos of varying intensities and in their early developmental stage, one may note the progressive deepening of the spectral colors as the lenticular clouding progresses, giving a beautiful kaleidoscopic appearance. Naturally, in the early stages these halos must be carefully regarded and glaucoma be excluded as an inciting cause. A well-developed halo may appear when looking at a light in the distance. According to the degree of illumination and distance at which lights are observed there can be a marked intensification of the cataract halo. At nighttime, on viewing the moon or stars, there may be reduplications and peculiar flask-appearance of a star.

Postaphakic Phenomena

Extracapsular extractions were performed on the left eye in a patient on November 21, 1949 and the right eye on June 5, 1950. A needling was done subsequently on the right eye approximately one year later (April 15, 1951). The slide shows the presence of a halo binocularly, but only monocularly in the right eye; also shown is the spectrum halo and prismatic effect of remaining lens capsule, and the spectral shadows the effect of numerous particles of lens material. Hazy vision prior to needling as well as these other phenomena illustrate the advantages of intracapsular extraction as a primary procedure.

Vitreous Opacities

One of the common complaints which bring patients to us frequently is the occurrence of black specks or spots before the eyes. These take all types and forms of shape, are usually innocuous but are of great concern to the individual. One of the conditions causing this is vitreous opacities. Opacities began suddenly on August 12, 1944 in a patient, and went through several periods of fluctuation, which at times were severe and alarming, but had cleared entirely some time prior to November 21, 1949 when the left eye was operated on. These opacities may develop in a rather compact form and spread out in irregular cobweb or lattice-like fashion with little dangling balls at the ends. A slide shows a late stage with gradual absorption of the vitreous opacities. Sometimes these opacities may form, or bunch together in clusters and take the irregular shape of distorted letters, particularly an S, L, or O.

Entoptic Phenomena

Entoptic imagery was brought into the realms of exact optical theory as the work

primarily of Purkinje, Listing, and Brewster.[11] It has to do with any object in front of the retina which is less transparent or has a refractivity different from the media in which it lies. The only type of entoptic phenomena we deal with here are those due to vitreous opacities or associated with blood corpuscles. The slides show various corpuscular-like bodies, mobile and of varying hues of pure white, pink, red, blue, green, and golden, as well as numerous fine short curved blue-black rods. Another type appears as red apple-like bodies in the green foliage of neighboring trees, also numerous small white mobile bodies as seen by the normal eye when gazing into the blue sky.

Peripheral Light Flashes

Here we come to one of the most interesting, unusual, and least documented phenomenon in the entire field of entoptic imagery.

Four distinct types of this phenomenon have been observed.

Type 1. On movement of the eyeball, brilliant white lines move along the margin of the visual field, especially when the movement of the eye is in the horizontal or vertical direction. These flashes will be at right angles to the direction of the movement.

When the movement of the eye is from below upward, the white flash starts at the left and shoots along the inferior periphery toward the right side. When the eyeball moved downward, the white flash begins at the left and moves along the upper edge toward the right. When the eyeball is moved from the left to the right, the white flash will move from above downward along the left periphery. When the eyeball is moved from right to left, the white flash will start at the upper right margin and shoot downward along the right periphery. These flashes of light occur chiefly when the eyeball is moved suddenly from a resting position. Vision is not disturbed by this phenomenon.

Type 2. This form, which perhaps is an independent manifestation, is a broad flash of light originating in the lower temporal quadrant and extending upward and slightly above the horizontal line, disappearing at the temporal side of the visual field. This phenomenon is not bilateral, does not recur nor is repeated, and has been observed on the mesial or nasal field of vision—there is no dancing or flimmering—just a broad white flash in one eye only. It may occur on the right side one day and the left side the next, or alternate on the same day. It does not interfere with direct vision. It usually occurs after retiring. There is no objective sensation, and it is not a part of the peripheral streak phenomenon.

Type 3. The term *scotoma albescens versare* (A.E.M., Sr.) has been given to this phenomenon because of the figures of rapidly moving flashes of light around the point of fixation. This type involves the central portion of the visual field; the peripheral field is not involved, remaining clear. The form of these figures varies greatly, but they all move in a circular manner surrounding the point of fixation, greatly interfering with vision. These figures vary in intensity and detail; their shape at times resembles the four blades of a ceiling fan in rapid motion. At other times the light flashes present the outline of the curved blades of the ordinary desk fan. Frequently the light flashes show as full blades instead of the outline of the edge. In others the figure presents a series of curved white lines radiating from the fixed central point whirling rapidly. Other figures consist of white circles of varying width, rapidly whirling around the central point. Some figures are made up of much broader elongated elliptical flashes encircling the central area with at times two or more circular figures occupying the central portion surrounded by whirling flashes of light. In some figures the movement of these flashing bands of light move clockwise and others counterclockwise. It is to be emphasized that these flashes of light occupy the central portion of the visual field and are of circular or whirling type in contradistinction to the movement observed in scotoma scintillans. The flashes of light are of very short duration, suddenly flashing in and out, about 3 times a second. Occasionally a figure of scotoma scintillans may occur simultaneously with this phenomenon. This visual disturbance develops on and after physical exertion such as climbing stairs, and is not dependent on the presence or development of crops of vitreous opacities. These flashes of light are perceived by, or are visible to the sufferer with the eyes open or closed. The frequency of the flimmer

is increased on suddenly opening the eye after firm closure. This phenomenon lasts only a few minutes and is associated with no headache, nausea, or paresthesia. Deep breathing, holding the breath, or straining does not change the rate of the "inward out" flimmer or the speed of the whirling elements of the figure. While conspicuous changes in blood pressure and frequency of the pulse have not been observed, a drop in pulse pressure may initiate this phenomenon.

Type 4. This type consists of the appearance of an agitated mixture of flashes of light at the margin of the visual field with the development of numerous flickering rings or circles of light "flickering" in and out throughout the visual field. At times these light flashes present thin circles; at others they contain waves of shimmering light which begin at the inferior margin and gradually fill most of the visual field, leaving a relatively clear area around the point of fixation.

This phenomenon depends upon the physical condition of the sufferer, is not accompanied by pain or headache, and is a transitory thing associated with a marked nutritional anemia. In one patient the RBC. count was 2,940,000, Hgb. 59.3%, and hematocrit 30%. With improvement in the RBC. count and Hgb., and with stabilization of the circulation, this phenomenon ceased and has not reappeared.

Summary

1. A detailed report on the visual phenomena experienced by one of us (A.E.M., Sr.) is presented. This includes migraine, scotoma scintillans, entoptic visualization of the optic nerve and retinal nerve fibers, early lenticular changes and postaphakic phenomena, as well as vitreous opacities.

2. Peripheral light flashes are covered in detail, listing four distinct types. To our knowledge these have not been presented in detail previously. To Type 3 has been given a name, *scotoma albescens versare,* a term chosen to describe the nature of the phenomena. Type 4 is a distinct clinical entity associated with a marked nutritional anemia.

3. Seventy-two Kodachrome slides are shown to illustrate the varying stages of these phenomena.

4. While phenomena discussed here are all of a subjective nature, it is hoped that their presentation will broaden our appreciation of the variety of these symptoms which our patients may present from time to time and help us visualize more fully their vague descriptions.

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Discussion (Abstract)

Dr. J. J. Stokes, Atlanta, Ga. Dr. Meisenbach has carefully recorded the entoptic phenomena which he observed over a period of 50 years. Their presentation was made even more interesting by drawings of the various phenomena.

Entoptic phenomena are, to most ophthalmologists, items of physiologic curiosity. However, as shown by Priestley and Foree (*A.M.A. Arch. Ophth.* 53:390, 1955), Scheerer's phenomenon and Haidinger's brushes are two of the entoptic phenomena which have advanced from experimental investigation to clinical application. Scheerer's phenomenon is present, minified, or absent in direct proportion to macular circulation. Haidinger's brushes appear or disappear in connection with edema of the macula. Variations in these phenomena may be elicited prior to the onset of ophthalmoscopically visible changes.

Dr. Meisenbach's presentation should increase the interest of ophthalmologists in the entoptic phenomena and we are indebted to him for an excellent presentation of a difficult subject.

Adverse Effects of Sunlight on the Skin

CAPT. JERE D. GUIN, USAF (MC), *Sheppard Air Force Base, Tex.*, and JOHN M. KNOX, M.D.,† *Houston, Tex.*

The fetish of sun-bathing to attain a sun tan is not without hazard to the lightly pigmented Caucasian. The untoward effects of excessive exposure to sunlight have received deserving attention in this paper.

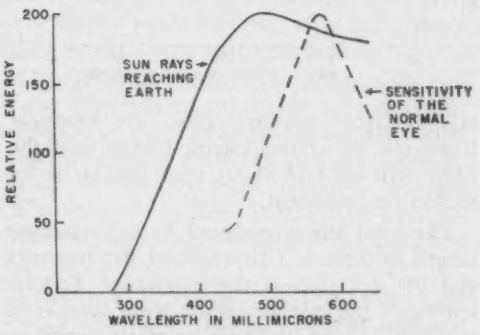
THERE ARE MANY DETRIMENTAL EFFECTS of sunlight which, until recently, have received too little publicity. Dermatologists in Texas have become acutely aware of the harmful effects which the sun may have on the skin because of the high incidence there of skin cancer and other diseases precipitated by sunlight. The practicing physician is often questioned by patients about sun-bathing, tanning, sunburning, sunscreening agents, and the like. In view of this concern, it is hoped this paper will enable the reader to understand better some of the more important aspects of the effects of sunlight on the skin.

Sunlight

Sunlight may be regarded as a continuous band of energy in the form of electromagnetic radiation. The sunlight spectrum at the earth's surface has components that range from 290 to 1,850 mμ (millimicrons) in wave length.[1] Visible light ranges from 390 mμ to 740mμ.[1] Infrared and ultraviolet light are found above and below the limits of visible light respectively (Fig. 1).

Almost all of the erythemogenic energy from ultraviolet light is absorbed by the skin, and very little light energy penetrates beyond a few millimeters.[2] The stratum corneum reduces intensity by scattering and absorption. Melanin absorbs ultraviolet light in the sunburn range, and since many tiny granules of this substance occur in the horny layer of darker-skinned individuals, these people react much less to exposure. The erythemogenic rays which penetrate the horny layer are absorbed in the malpighian layer by molecules of protein and/or nucleoprotein. The light energy raises these molecules to a higher energy level, and chemical and biologic changes occur.

FIG. 1



Responses of Normal Skin to Sunlight

I. *Erythema.*

Erythema is produced by a well-defined range of sunlight between 290 and 317 mμ in wave length. The greatest effect occurs at 297 mμ. Erythema may also be produced by artificial sources such as the cold quartz and the hot quartz ultraviolet lamps. The cold quartz ultraviolet lamp, with a peak effect at 254 mμ, produces an erythema which is not followed by pigmentation. Severe burns may be produced by intensive or prolonged exposure to erythemogenic ultraviolet light. Many systemic effects have also been noted, and these are summarized in Ellinger's book[3] (Fig. 2).

The exact process by which ultraviolet light causes reactions in the skin is not known. Probably the injured cells liberate leukotaxine and other substances which induce inflammation.[2] Sunburn may be prevented by a filter or a sunscreen which absorbs or scatters most of the ultraviolet light below 320

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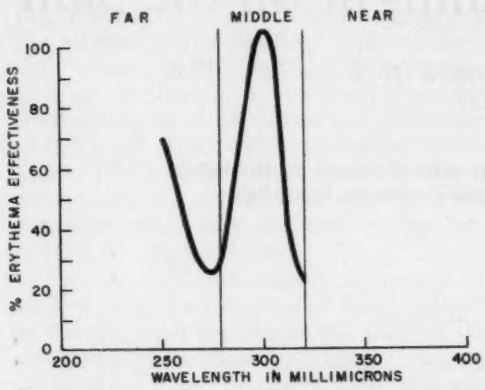
FIG. 2



mμ. Ordinary window glass, for example, filters out the erythemogenic rays of sunlight. More will be said about such agents in the section on treatment.

The total effect produced by a given wave length of light is a function of the intensity and the duration of the exposure.[2] The intensity of the erythemogenic wave lengths of sunlight, in turn, depends on many variables. The amount of sunburn producing radiation in sunlight is much greater at noon than it is in the early morning or the late afternoon. It is also much higher in the summer than it is in the winter. Erythemogenic radiation is more intense at tropical latitudes and at higher altitudes if other variables are the same.[4,5] Reflected light from the environment also tends to increase the total exposure.[6] Water vapor and dust particles decrease erythemogenic radiation. The normal limit of ultraviolet light from sunlight at 290 mμ is caused largely by absorption by ozone of shorter wave lengths[5] (Fig. 3).

There are also many variables in the response of the individual to a given quantity of erythemogenic ultraviolet light. The face and arms respond less than the abdomen or back in the same person.[2] Dark-complexioned people are less responsive than light-skinned ones. Those who have been chronically exposed to sunlight may develop a degree of tolerance thereto. Protection is provided by the horny layer and its pigment granules,[6,7] and to some extent by sweat.[8]

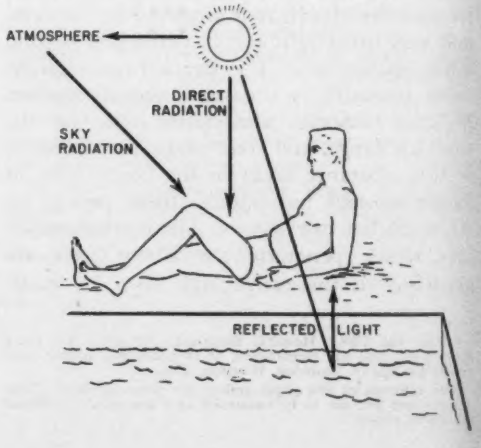
II. *Tanning.*

Sun tanning involves three basic processes. Two of these are changes in melanin already present, and the third is the formation of new melanin. The melanin present is oxidized to a darker state following exposure of the skin to ultraviolet light. The greatest stimulus for this process results from wave lengths of about 300 to 420 mμ, with a peak at about 340 mμ.[9] The second reaction is an upward dispersion of melanin granules in the epidermis which occurs several days after irradiation.

Formation of new melanin is caused by wave lengths of ultraviolet light between 280 and 310 mμ. Melanogenesis begins about 2 days after exposure to ultraviolet light, but the height of pigment production is not reached until at about 19 days. The amount of pigment begins to decline after about one month and is back to normal in approximately 9½ months in the absence of any further exposure.[10] The process of melanin formation starts with tyrosine, which is metabolized in the melanocyte to dihydroxyphenylalanine (DOPA) by the copper-containing enzyme tyrosinase. From DOPA the process goes through several metabolic stages until indole 5,6-quinone is formed, and apparently this is polymerized to form melanin.[10] The stimulation of melanogenesis by ultraviolet light has been explained as due to a shift of S-H bonds to disulfides, direct catalyzation of tyrosine to DOPA, a lowering of the redox potential, and the increase in skin temperature following ultraviolet light exposure which accelerates the conversion.[11]

Freckling is a peculiar type of tanning

FIG. 3



which seems to be linked to the gene determining red hair.[12] Here the number of melanocytes is unchanged, or actually decreased,[13] but the production of pigment is increased in local areas causing a localized increase in the skin content. This process is stimulated by sunlight in about the same manner as normal tanning.

III. *Senile skin, keratoses, and epitheliomas.*

Following prolonged exposure to sunlight, the skin tends to show premature aging. A dramatic difference can often be demonstrated, especially in fair-complexioned persons, by comparing the skin above and below the collar line. This degenerated skin above the collar line is sometimes called senile elastosis. The fashionable women who regard a "beautiful sun tan" essential would do well to consider the long-term results.

Actinic degeneration (farmer or chataigne type skin), keratoses, and epitheliomas are also seen, especially in fair-skinned individuals following chronic exposure to sunlight. These conditions are found more frequently in the South and Southwest than in the North of the United States. Severe "farmer skin" or "chataigne skin" is frequently found in outdoor workers of southwest Texas. Such individuals tend to develop multiple skin tumors. A histologic section of "chataigne" skin will show mild to moderate atrophy with a degeneration of the collagen in the upper cutis.

Senile keratoses are clinically hyperkeratotic lesions with little or no erythema or induration. Histologically, a senile keratosis is a squamous cell carcinoma, Grade ½; i.e., malignant cells are seen, but there is no invasion. This lesion may progress and become invasive, however, and it is then called a squamous cell carcinoma.

Epitheliomas of the basal cell and squamous cell types are found especially in blue-eyed blonds or redheads who have been chronically exposed to sunlight. Farmers, sailors, policemen, and other outdoor workers are especially prone to develop these lesions. The rarity of carcinoma of the lip in women who wear lipstick and the rarity of cutaneous malignancy in Negroes demonstrate the benefits which may be obtained by protecting the skin from the sun. Since the albino Negro does not have this protection, he is especially prone to develop actinic degeneration and keratoses.

IV. *Other changes in the skin following sunlight exposure.*

The epidermis responds to sunlight with an increased thickness of the stratum corneum which tends to afford some extra protection.[1] Another action of ultraviolet light on the skin is the conversion of provitamin D to vitamin D. This was believed to occur in the surface lipid layer so that vitamin D was absorbed through the hair follicles. However, more recent work indicates that the change occurs in the upper malpighian layer.[14]

Changes occur in the skin amino acids following exposure to ultraviolet light and with oxidation of tissue proteins. Abnormally high values of certain amino acids have been reported in various light sensitive diseases. These values have been reversed by administration of BAL, supposedly because BAL acts as a reducing agent.[15]

Subsequent to exposure to ultraviolet light there is a systemic vascular response followed by a localized vascular response in the irradiated area of the skin. An increased blood flow lowers both the systolic and diastolic blood pressure. Some individuals with active pulmonary tuberculosis have experienced hemorrhage from pulmonary lesions following excessive sunlight.[10]

Light Sensitivity and Light Induced Diseases

I. *Photo-allergy, phototoxicity, and photodynamic action.*

Photo-allergic diseases belong to the broad group of physical allergies. Baer and Sulzberger's theory[16] probably best explains the mechanism. Following exposure to a physical agent, certain metabolites are normally liberated. Some few individuals have the capacity to become allergic to these metabolites which occur in irradiated normal skin, while the great majority do not; and photo-allergy occurs only in the few who form antibodies to these metabolites. Many such cases have definitely been proven to be allergic by passive transfer technic. Other cases must be surmised to be allergic because of circumstantial evidence, e.g.: (1) the presence of an incubation period; (2) reaction in previously exposed areas distant from the irradiated area; and

(3) reproduction of lesions by using nonspecific radiation, e.g., alpha and gamma rays.

Solar urticaria is a photosensitive disease in which the patient develops hives following sunlight exposure. These patients are sensitive to one of the following four zones of light: (1) below 310 mμ, (2) below 370 mμ, (3) 400 to 500 mμ, or (4) infrared.[16] In many cases it is possible to transfer the sensitivity passively. Often the reverse passive transfer test is also positive, and this suggests that the allergen is a normal metabolite. (In the reverse PK test, the patient's serum is injected *after* exposing the normal skin to light. Thus, the antigen responsible for the reaction is formed by normal skin, and it is therefore a normal metabolite.)

Phototoxicity, according to Epstein,[17] is the primary nonallergic type of photosensitivity based on the pharmacologic action of a sensitizing substance. This type of reaction occurs in normal individuals and it varies quantitatively in proportion to the dosage. A photochemical reaction is probably the chief factor here. Photodynamic action, sunburn reaction, and primary sulfanilamide response are cited as examples of phototoxic reaction.

Photodynamic action is a type of photochemical reaction which requires the presence of a photosensitizing agent, light of the proper wave length, and molecular oxygen. This photosensitizer absorbs light energy in a specific range and it essentially acts as a catalyst. Blum[1] postulated the mechanism of this reaction to be as follows. Light is captured by the photosensitizer, thus raising its molecules to a higher energy level. The excess energy is transferred to the substrate which, in turn, reacts with oxygen. Oxidation of the substrate produces damage to the cellular structure. The photosensitizer is unchanged, however, and it is ready to accept more light energy of the proper wave length.

Individuals into whom rose bengal is introduced can be shown to be photosensitive to light with a peak effect at about 550 mμ. Here, visible light causes whealing in areas of exposure. This reaction is photodynamic and will not take place in the absence of oxygen.[18] Other photosensitizing agents include certain perfumes, meadow grass, phenothiazine, coal tar, figs, limes, parsley, and many others.[12] The highly publicized tanning agent, 8-methoxypsoralen, is also photodynamically active. Photodynamic action does not depend on an antigen-antibody mechanism, as does photo-allergy. Photo-allergic reaction may follow photodynamic action, however. Sams[19] noted a delayed urticarial reaction following the application of Persian lime oil and exposure to sunlight. This reaction occurred about a week after exposure, while the ordinary erythema due to photodynamic action was noted at 24 hours. This did not occur in all of the individuals tested, but probably only in those who were capable of forming antibodies to the liberated metabolites. Thus, the development of a photo-allergic reaction depends upon the individual's ability to form antibodies to normal metabolites, and the photosensitizing substance is merely involved in the liberation of these metabolites.

II. *Porphyria.*

Porphyria may be divided into two main categories, erythropoietic and hepatic. The latter includes acute intermittent porphyria and porphyria cutanea tarda. The most sensitive of these to sunlight is the congenital type (erythropoietic), and the least photosensitive is the acute intermittent. The major absorption of the porphyrins as a group lies between 300 and 650 mμ. Major absorption bands occur between 300 and 410 mμ and 506 and 602 mμ, depending on the type of porphyrin.[7] Uroporphyrin-I is the chief porphyrin found in congenital porphyria. It has a peak absorption *in vitro* near 400 mμ. Lesions are not readily reproduced by light of the 400 mμ wave length, however, and this may be because the absorber *in vivo* is a protein porphyrin complex.[20]

Porphyria cutanea tarda usually manifests a bullous eruption, especially over the dorsum of the hands. Histologically, these lesions are subepidermal bullae with a relatively small amount of inflammatory reaction in the surrounding area. The bullae are precipitated by trauma similar to epidermolysis bullosa, but the underlying defect probably is caused by a photodynamic reaction since light exposed areas are almost exclusively involved.

III. *Koebner phenomenon.*

There are many skin diseases which may show the Koebner phenomenon, i.e., the tendency to form lesions of the disease in a site of trauma. Sunlight can produce such a

response. The sunlight-induced skin lesions of pellagra belong in this category, and other diseases, such as psoriasis, lichen planus, pityriasis rubra pilaris, and Darier's disease, may show this.[7]

Lupus erythematosus may be classified as acute disseminated, subacute disseminated, and chronic discoid. It has been shown that the discoid and disseminated varieties are probably related although some doubt this.[12] Since most physicians are familiar with the findings in these conditions, a clinical description will not be given. The skin findings in this disease have often been shown to be adversely influenced by sunlight, and they are usually found on light-exposed areas. Also, exposure to sunlight may produce severe exacerbations of lupus erythematosus.[19] Recent tests with fluorescein-tagged antibodies indicate the presence of a sensitivity to DNA. protein.

IV. *Xeroderma pigmentosa.*

Xeroderma pigmentosa is a rare hereditary disease characterized by an extreme exaggeration of the individual's response to sunlight. Clinically, these patients develop hyperpigmentation, atrophy, and telangiectasis. The skin undergoes premature senile (actinic) degeneration and cutaneous malignancies develop early in life. Protection from sunlight is extremely important in the management of this disease.

V. *Polymorphic light eruption and hydroa aestivale.*

According to one classification, the term "polymorphic light-sensitive eruption" includes four morphologic variants: (1) plaque-like type, (2) contact eczematous type, (3) papular and prurigo-like type, and (4) erythematous type (erythema perstans solare). The condition is seen chiefly in the summer with remissions during the winter. While it is usually seen only on exposed areas, one side may be involved to a greater extent than the other. Not infrequently automobile drivers will have more extensive involvement on the left side, and those who are riders will have more on the right side. The pathogenicity of this condition is not well worked out. However, it has been shown that it is possible to transfer hypersensitivity to light passively in some patients with the papular and prurigo-like type (prurigo aestivale). Therefore, one can surmise that at least some of these cases are due to a photo-allergic mechanism. It has been reported that a low normal excretion of 17-ketosteroids, low sperm count and creatinuria occur in these patients. The skin eruption in most patients responds to testosterone or gonadotropin therapy. However, the antimalarial drugs will control the eruption more rapidly, and appear to be the treatment of choice.

Hydroa aestivale is most often seen in children as a vesicular eruption over the face and the lateral aspect of the arms. It usually spares the area under the chin and along the medial aspect of the arms. It tends to occur in the summer and recede during the winter, and it frequently clears spontaneously at about puberty. Occasionally it is associated with porphyria of the congenital variety, and in these patients the eruption does not ordinarily subside at puberty.

VI. *Other light-induced dermatoses.*

While there are some rare light-induced diseases not included above, the one last entity which will be discussed here is colloid milium. This occurs primarily in fair-skinned people who are chronically exposed to intense sunlight. The incidence is much higher in Texas than in the northern United States, and it was seen with increased frequency in the East Indies during World War II. The lesions are pseudovesicles containing colloid material of blood protein origin. They are seen most frequently over the dorsum of the hands, the face, and the neck. The only satisfactory treatment is the avoidance of sunlight.

Protection from Sunlight

I. *Natural protection.*

The natural protection of the skin lies chiefly in three factors: (1) the thickness of the stratum corneum, (2) the amount of pigment present in the epidermis, and (3) the surface sweat. The stratum corneum tends to scatter and absorb erythemogenic light. Exposure of the skin to sunlight is followed by a thickening of this layer which affords an increase in protection. The stratum corneum of dark-skinned individuals contains numerous melanin granules. This layer in the Negro offers several times as much protection

as the same layer in the Caucasian or in the albino Negro. Pigment in the basal layer is thought to offer very little protection since damage from sunlight occurs above this level. It is possible, however, that melanin may be active chemically as well as optically, in which case it would be active throughout the epidermis. Sweat also absorbs some ultraviolet radiation, chiefly due to the urocanic acid present.

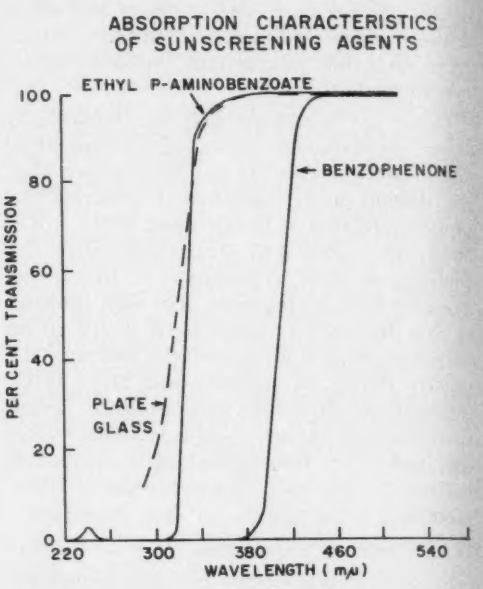
II. *Ultraviolet light filters.*

Window glass is a good filter of the sun's rays which produce sunburn.[1] Since it does not filter out the near ultraviolet radiation, it is not adequate in the protection of patients with sensitivities above 320 mμ.

Chemical sunscreens are agents that protect the skin by absorbing damaging ultraviolet light rays and dissipating their energy in some harmless manner. Organic compounds which have absorbed radiation are either raised to a higher energy level or dissociated. The excited molecules may dissipate their absorbed energy by collision, fluorescence, or a reaction with other molecules at collision.[1] Probably the most commonly used sunscreens today are para-aminobenzoic acid (PABA.) and its esters. These products are excellent sunscreens in the middle ultraviolet range where sunburn-causing rays are found. However, they do not affect the near ultraviolet range. Recently, we reported a new group of sunscreens which are at least as good as, if not better than, PABA. in preventing erythema. In addition, these compounds have a much wider absorption spectrum than PABA., and this fact may make them better agents in treating diseases caused by the near ultraviolet radiation. Since permanent tanning is initiated by a wave length of light very close to that of sunburn, one should not expect melanin formation with high concentrations of PABA. or benzophenones. We have found most commercial suntanning preparations to be only 3 to 5% as effective in screening erythemogenic rays as 10% concentrations of PABA. or certain benzophenones in vanishing cream (Fig. 4).

Physical sunscreens (sunshades) are opaque chemicals that scatter light rather than absorb it. Examples are titanium dioxide, talc, kaolin, zinc oxide, and betonite. These are used at times to complement the protection afforded by chemical sunscreens. Certain substances used as vehicles also absorb erythemogenic radiation and provide a limited amount of protection from sunburn. Yellow petrolatum, for instance, offers fairly good protection.[6,7]

FIG. 4



III. *Systemic agents.*

Much research has been done recently on a derivative of the *Ammi majus* plant known as 8-methoxypsoralen. This particular product promotes tanning by a photodynamic action. There are some inherent drawbacks in the use of this product, however. First and foremost, the skin not only tans more readily, but it also sunburns more easily. Extreme care must be taken in the use of these products, both orally and topically, to avoid the sunburn reaction. Again, on discontinuing the use of these agents, the melanin concentration of the skin will decline; therefore, the agents must be readministered at regular intervals. 8-MP may also be hepatotoxic, although this has recently been questioned.

In recent work with mice, this agent, given orally, has been shown to decrease slightly the erythematous and carcinogenic effect of hot quartz ultraviolet light. However, it produces a carcinogenic effect in mice with the longer wave lengths of the Wood's lamp spec-

tra, and even with fluorescent light. It is highly doubtful that 8-methoxypsoralen will ever be a valuable tool in the treatment of light-caused dermatoses.

The antimalarial drugs are probably the best known treatment for discoid lupus erythematosus and polymorphic light eruptions. Often dramatic results are obtained with a daily dose of only one 250 mg. tablet of chloroquine. At times, one antimalarial preparation seems to be more effective than the others, and oftentimes when progress with one antimalarial drug has ceased, considerable benefit may be obtained with another.

The exact mode of action of the antimalarials is not known. Some studies deny that it is due to their sunscreening effect, whereas other investigators have stated that the action of quinacrine and chloroquine may be due to their effect on flavin enzymes. They state that quinacrine inhibits D-amino acid oxidase and cytochrome reductase. It also promotes excretion of riboflavin and growth of young rats fed suboptimal amounts of the vitamin. Quinacrine thus may partially replace riboflavin in the flavin enzymes. It also may inhibit destruction of riboflavin or one of its conjugates. An effect on DNA. depolymerization has also been proposed. The antimalarial drugs probably have no effect on sunburn.

Certain light-induced dermatoses, e.g., polymorphic light eruption, actinic keratoses, and epitheliomas, are much less common in heavily pigmented races. It would be interesting, therefore, to see what effect melanocyte-stimulating hormone (MSH.) would have on these diseases. This hormone is very hard to obtain at present, and research along this line will have to await the time when MSH. is more readily available.

Summary

1. Sunlight is the precipitating factor of a large family of diseases including: sunburn, senile keratosis, skin cancer, premature aging of the skin, and a group of light sensitive and light induced diseases. The mechanism of production of these various conditions is briefly discussed.

2. Photo-allergy, phototoxicity and photodynamic action are defined and briefly described.

3. Various methods of protecting the skin from the harmful effects of sunlight are presented. These include the natural protection of the skin, chemical and physical sunscreens, and various systemic agents.

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The authors offer further references upon request.

Extrathoracic Aortic Shunt: An Experimental Technic

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TEMPORARY AND REMOVABLE AORTIC SHUNTS have been utilized to allow the flow of blood where minimal interruption of the circulation is mandatory. Izant and associates[1] and Hurwitt[2] have reviewed the development and types of shunts. These have been hollow internal prostheses either porous or nonporous, rigid or nonrigid.

To test an external aortic shunt secured in a vessel by a new form of anastomosis, and to test the feasibility of preparing a short-term experiment in an animal, wherein the entire blood supply of the descending thoracic aorta passed outside the body, the following experiments were performed.

Five mongrel dogs varying between 8.8 and 14.7 kg. were anesthetized with 5.5 mg./kg. of intravenous Nembutal and attached by means of an endotracheal tube to a mechanical respirator. With aseptic technic the chest was entered through the left 4th or 5th interspace and several pairs of intercostal vessels ligated and divided. Semirigid vinyl plastic tubing of varying sizes was then inserted in the proximal aorta by a double suture ligature. Care was taken to barely approximate the vessel to the prosthesis, so the circumferential ligatures would not cut through the vessel wall. This tube was brought out through a small stab wound at the 7th or 8th interspace as far posteriorly as possible. (This is an area which is difficult for the animal to reach with either its feet or mouth.) The distal anastomosis was then performed and tension of the vessels relieved by ligatures where the prosthesis crossed itself (Fig. 1).

Results. Four of the five animals were considered experimental successes in that they survived the immediate operative procedure. Three animals lived for 6 or 7 days and one animal for 3 days. Two of the anastomoses leaked, and one clotted. The cause of death could not be ascertained in the other animal.

FIG. 1

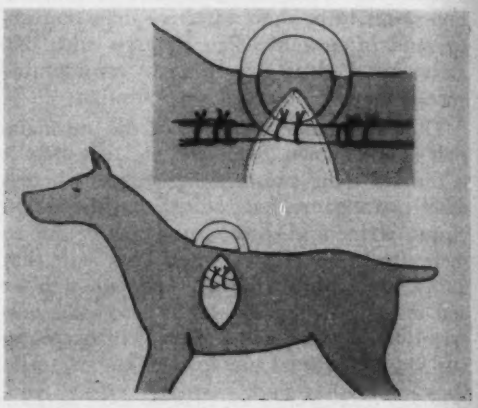

Diagram showing insertion of semirigid vinyl plastic tubing.

The animals which survived the immediate postoperative period were normal until just before death. Blood could be seen passing through the plastic tubing, and a pulse could be felt through the tube.

Discussion

A shunt of the type described meets the criteria of a satisfactory temporary aortic shunt as described by Izant and associates.[1] These are: (1) simplicity of design, (2) ease of introduction into and removal from the aorta, (3) sufficient lumen to permit adequate blood flow, and (4) enough security to prevent leakage and accidental dislodgment. In addition, this type of prosthesis functions well outside the body for moderately long periods of time, exceeding by days the time required of temporary shunts for present operations. With possible development of future vascular technics, such as long-term extracorporeal hearts and arterial infusions, arterial prostheses which can enter the circulation from outside the body may become important. A clue to the potential effectiveness of extracorporeal vessels has been furnished by Scharmel and Creech.[3] In their report fabric arterial grafts in man,

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which were exposed by infection, continued to function for months.

Summary

Experiments on 4 animals show that all the blood passing through the descending aorta can be diverted to the outside of the body in the dog by means of a plastic shunt for periods up to one week.

Acknowledgment

We would like to thank Mrs. Marsia Herman for her technical assistance.

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Improved Treatment of Infiltrating Bladder Tumors With Cobalt-60 in Nylon Sutures and P-32: A Co-ordinated Effort of Urologist, Pathologist, and Radiologist

J. R. MAXFIELD, JR., M.D., JACK G. S. MAXFIELD, M.D., VINCENT VERMOOTEN, M.D., and J. L. GOFORTH, M.D.,† *Dallas, Tex.*

The authors describe a technic of irradiation by the implantation of Co^{60} within the malignant tissue. The results appear good, and certainly without the complications associated with radical surgery and so unpleasant in the life of the patient.

CARCINOMA OF THE BLADDER, the most prevalent and important malignant tumor of the urinary tract, caused some 5,000 deaths in the United States last year. This represents about 3% of the total deaths from malignant disease. Although various improved technics have been developed during the past 20 years, the death rate has remained fairly constant.

A review of the literature on carcinoma of the urinary bladder emphasizes the universality and persistence of this problem. As early as 1851, Samuel D. Gross in his treatise on *Diseases of the Urinary Bladder* cited the first description of vesical papillomatosis given by Dr. John Green Crosse (1833), and several other instances of vesical neoplasm taken from European publications. During the ensuing years, numerous case reports, pathologic descriptions, and methods for treating these bladder growths have appeared in the medical literature. Following the demonstration by Beer, in 1910, that bladder tumors could be destroyed by fulguration, the urologist has regarded these tumors as his domain. However, the pathologist and the radiologist may also play important roles in the treatment of bladder tumors. The controversy continues to the present time with opinions concerning the best treatment still differing markedly.

In selecting the method of treatment, such factors as classification, extent, and radiosensitivity of the tumor must be determined. The pathologist supplies pertinent data concerning the nature of the lesion. Lockwood and Chapman[1] emphasize "to establish a diagnosis requires the exercising of the maximum of good judgment and scientific investigation." It is often difficult to estimate the degree of malignancy, extent of infiltration, and presence of metastasis on the gross cystoscopic appearance of the lesion alone. Even microscopic findings on the biopsy material can be misleading. If the specimen is of inadequate size, fails to include the base of the lesion, or is not a true cross section of the lesion, the malignant portion of a papillomatous tumor can be missed. This undoubtedly accounts for the report of a benign epithelioma that later is found to have malignant recurrence or extension. These errors occur because bladder lesions are characteristically papillomatous in nature with a mixture of widely dispersed clumps of malignant cells in a large field of benign tissue. Thus, a correlation of the findings from cystoscopy, biopsy, cystograms, P^{32} tracer, and bimanual palpation is required to define the lesion to be treated.

Broders, Colby, Friedman and Lewis, Jewett, Kirwin, and Marshall have published excellent descriptions and classifications of bladder tumors. They have considered the stage (anatomic extent), grade (histologic degree of malignancy), geometrical configuration (diameter and thickness), origin (single or multicentric), and occurrence (primary or recurrent).

For the series presented in this article, the

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following classification of urinary bladder tumors (pathologically and clinically) has been employed:

Group 1 is composed of benign papillomatous type tumors which are low grade, noninfiltrating, noninvasive, but often recurrent. Tumors of this type may evolve from single or multicentric origin in the vesical mucosa and tend to cover the entire bladder surface.

Group 2 includes the solitary, actively invasive, infiltrating carcinoma; sessile tumor; and superficially ulcerating squamous cell tumor. Any invasion of the tunica propria or muscularis by a bladder tumor places it in this group as long as it is still confined within the bladder wall and has not spread through it into the extravesical tissues.

Group 3 is comprised of tumors which have spread beyond the confines of the bladder wall and have invaded the perivesical tissues. Distant metastatic involvement may or may not be present.

As previously mentioned, the proper method of treatment for carcinoma of the urinary bladder has been under discussion and constant change since the discovery of man's ability to utilize surgery and radiation. We support the doctrine that treatment depends primarily on whether infiltration of the bladder wall has occurred and, if so, to what extent; and secondarily, on the evaluation of the location, multiplicity, degree of malignancy, and size of the growth.

Treatment procedures for Group 2 have ranged from extreme conservatism to radicalism both in surgery and radiation therapy. The value of any method of treatment depends primarily on its efficacy in totally eradicating the primary growth, but at the same time it should not cause unnecessary objectionable complications.

Well documented in the surgical and urologic literature are the various surgical procedures used for bladder tumors such as fulguration, electrocoagulation, electro-excision, segmental resection, total cystectomy, and total pelvic evisceration. In more recent years there has been a definite trend away from very radical surgery, particularly the total cystectomy approach. Segmental resection is also viewed with considerable caution unless it is reasonably certain that the operative procedure can reach well beyond the tumor margin, remove a wide cuff of healthy bladder wall, and still leave a functioning bladder.

Radiation therapy for bladder tumors may be divided into three major categories: external, intracavitary, and interstitial. Whether radiation therapy is to be the primary form of treatment or employed in conjunction with other therapeutic methods must be considered. Poor tolerance both systemic and by the bladder has discouraged external radiation to the whole bladder, using the standard low voltage or deep x-ray therapy machines. External roentgenotherapy with supervoltage and cobalt-60 teletherapy units has proved more effective as reported by Colby, Burkell and Watson, Buschke and Cantril, Cordonnier and Seaman. Goin[2] developed a low voltage contact irradiation procedure through a suprapubic wound into a marsupialized bladder, with good results. Rose[3] advocated direct irradiation of the tumor, giving one dose of 3,300 r.

In 1911, Paschkis,[4] followed by Pasteau and DeGrais,[5] Schüller,[6] and Hugh Young, devised apparatus for placing radium in the bladder. In 1922, Neill[7] implanted radon seeds into the bladder tumor through an endoscope. Dean,[8] Barringer,[9] Keyes,[10] and Watson[11] used transvesical implantation of radon seeds into the bladder wall, with good results. Wasson[12] and Friedman and Lewis[13] devised apparatus to place a radium source in the bladder by encasing it in a balloon catheter. Later, radioactive cobalt-60 pellets or isotopes in liquid form such as sodium-24, bromine-82, and gold-198 have been used in the same manner for intracavitary irradiation. Recently, radioactive solutions such as gold-198 have been instilled directly into the bladder by catheter as an alternative to total cystectomy.

In 1944, Wayman[14] implanted low intensity radium needles extravesically through the outer portion of the bladder wall and tumor. In 1951, Morton, Callendine, and Myers[15] reported the use of radioactive cobalt-60 in plastic tubing for interstitial radiation therapy of various areas in the body, including the urinary bladder. In 1952, Binnie and Bates[16] used interstitial implantation of curved rigid needles containing radioactive cobalt into the bladder tumor. Wallace, Stapleton, and Turner,[17] Sinclair,[18] Smithers,[19] Van Miert and Fowler[20] implanted radioactive tantalum wire interstitially from within the bladder lumen

and also from the bladder's outer aspect in association with partial cystectomy, with good results.

Co^{60}-Nylon Suture Technic

From the excellent results obtained by Morton and associates, the use of radioactive cobalt-60 in Nylon tubing seemed an ideal method for treating Group 2 bladder tumors and was first applied by us in 1952. The technic proved versatile in that the radioactive cobalt-60 in hollow Nylon sutures could conform to the bladder contour and be placed in a carefully planned implant without significant radiation to the operating team. This was accomplished by keeping the radioactive cobalt-60 in the lead container on the Mayo table while placing the Nylon suture leaders and by finally pulling the radioactive material into place after the placement pattern had been completed. The results obtained by this technic were encouraging, and a series of cases was undertaken using radioactive cobalt-60 in Nylon sutures as the basic therapy.

To aid in evaluating the tumor, 24 hours or more prior to the radioactive cobalt-60 implantation procedure, the patient is given a tracer dose of 1,000 microcuries of radioactive phosphorus intravenously. At the time of operation the tissues removed are checked for radioactivity. Areas containing tumor show an increased localization of the radioactive phosphorus. Autoradiographs are made on all the tissues removed surgically and carefully compared with the pathologist's findings.

The operating team is ideally composed of a radiologist and a urologist, since both specialists are necessary to secure an implant that is the best possible, both radiologically and surgically.

The operating team exposes the bladder through a suprapubic incision. The bladder is opened and the involved wall is freed so the actual extent of the tumor can be palpated as well as visualized. If the tumor measures more than 1 cm. in thickness or protrudes into the bladder cavity, it is thinned down with a high frequency cutting loop to about the level of the bladder wall. The long nonradioactive ends of the Nylon sutures are placed to form a planar implant employing as many sutures as necessary to effect a satisfactory radiation pattern. To obtain a proper implant the sutures should be spaced approximately parallel about 1 cm. apart. By having the outside wall of the bladder exposed, members of the operating team can feel the bladder wall between their fingers and make the placement of the interstitial cobalt in Nylon sutures with a high degree of accuracy. Each Nylon leader is coded for proper identification to facilitate any necessary shifting or removal to achieve the desired radiation dosage to the area. The first suture should be placed 0.5 cm. beyond the outside edge of the visible or palpable tumor. It is preferable to have the margins of the radioactive material in normal bladder tissue. After all of the nonradioactive leaders are sutured in the desired pattern, the portions containing the tiny sealed-in slugs of radioactive cobalt-60, separated by aluminum spacers, are pulled from the lead safe into their place in the implant. Only then is the operating team briefly exposed to radiation. Radiation exposure of the operating team is thus kept at a minimum by this technic.

While the patient is still on the operating table, radiographs are made in the anteroposterior and lateral projections. If the placement pattern demonstrates that additional sutures are needed or that the radioactive sources are improperly positioned, this can be easily corrected at this time.

The bladder and abdominal wall are then closed, and the coded Nylon sutures are brought out through stab wounds 2 or 3 cm. on both sides of the incision.

To help prevent generalized cystitis, the opposite bladder wall is kept at least 4 cm. away from the implant by inserting a 30 cc. balloon indwelling catheter. The balloon is filled with water to protect the opposite bladder wall and to help maintain the position of the radioactive cobalt-60 in the bladder wall.

Ordinarily the radiation pattern is planned so the radioactive cobalt-60 will remain in place 7 or 8 days. The exact time of removal is determined by calculating the number of hours necessary for the finally achieved pattern to deliver a dosage of 7,000 gamma roentgens to the tumor area in the bladder wall. From 48 to 72 hours after operation, radiographs are made in stereoscopic anteroposterior and lateral projections. From these films a three dimensional reconstruction of the implant is made. If indicated, any necessary shifting of the sutures can be carried out. Employing the three dimensional reconstruction of

the final radioactive cobalt pattern and appropriate Paterson-Parker tables, the time in hours required to deliver the desired dosage of 7,000 gamma roentgens can then be calculated.

When the desired radiation dosage has been attained, the Nylon sutures containing the radioactive cobalt are removed by simple extraction in the same manner as through and through steel sutures. Usually, the patient is given intravenous Nisentil for the removal procedure in the operating room, but occasionally, Sodium Pentothal may be necessary. Care must be taken to insure that all the radioactive portions of the sutures are removed and completely redeposited within the lead safe.

Following this procedure, observational examinations are made at 6 weeks, 3 months, 6 months, and yearly intervals. These include general physical examination, excretory urograms, cystoscopic examination with determination of bladder capacity, blood count, urinalysis, and Papanicolaou stain of the urinary sediment. The 6 months examination should also include tumor survey radiographs for detection of possible metastasis.

Should the interval examinations demonstrate recurrence of the bladder tumor, the cobalt-60 in Nylon procedure can be repeated by reopening the bladder and carrying out the implantation again. It would rarely be necessary to consider repeating this technic earlier than 4 to 6 months.

Results

From November 1952 to September 1956, 22 patients with an infiltrating bladder carcinoma (Group 2) have been treated with the above technic. Of this group, 1 died from myocardial infarction 48 hours after operation, 1 died from pneumonia 6 weeks following operation, and 3 died of metastatic carcinoma to the liver and elsewhere. The remaining 17 patients are alive and are clinically and cystoscopically free of tumor. It has been 5½ years since the first cobalt implantation and 5 years since the second implantation in the initial patient. It has been 5 years since cobalt implantation for 2 patients, 4 years for 5 patients, 3 years for 3 patients, and 1½ years for 6 patients.

Summary

Through the teamwork of the urologist, pathologist, and radiologist, interstitial radioactive cobalt in Nylon sutures offers these advantages in treating infiltrating bladder tumors (Group 2 as above described). It delivers to a predetermined site in the wall of the bladder a carcinocidal dosage of 7,000 gamma roentgens or more, with minimal irradiation exposure to the opposite wall and remainder of the bladder. The implant pattern can be tailored to fit the tumor and positioned accurately to achieve uniform radiation dosage. By shifting the pattern or removing some of the sutures, early "hot spots" can be prevented without difficulty. By leaving the coded ends of the sutures on the outside of the body, a change in the pattern can be accomplished by merely adjusting the sutures. Radiation exposure to the operating team is kept at a minimum.

The advantages of this technic for the patient include the preservation of satisfactory bladder function without incontinence, and the elimination of various problems associated with the operations for ureteral diversion. In several cases where the ureter has been obstructed by tumor at the ureteral orifice, the kidney on the involved side has been salvaged. It offers a high percentage of cures; but if a cure cannot be obtained, prolongation of life and excellent palliative results are usually achieved.

Thus, the combination of radioactive phosphorus as a diagnostic aid and interstitial implantation of radioactive cobalt-60 in Nylon sutures as treatment for infiltrating bladder tumors causes less deleterious consequences than other types of irradiation and surgical procedures, leaves a more normally functioning urinary bladder, requires no "device" to collect the urine, and offers an excellent chance of cure.

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CASE REPORT

The Treatment of Poisoning in Children by Exchange Transfusion:

A Review of the Literature and Report of a Case*

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DEATH DUE TO ACCIDENTAL POISONING is very common in young children, drugs being the cause in about one third of the deaths. One third of the latter are due to the ingestion of aspirin and salicylates. These drugs are so accessible and common that their part in accidental mortality and morbidity is easily understood.[1]

Axtrup[2] described in detail, in 1950, exchange transfusion as a treatment in poisoning. Among his most interesting cases were two, a 4 year old girl who had swallowed chloramine-T tablets, and a 2 year old girl who had swallowed an unknown quantity of marking ink, which consisted of a mixture of nigrosine, a very poisonous aniline dye, aniline oil and benzene. He believes that exchange transfusion should be used only as a last resort, and should be used primarily in poisoning due to the aniline preparations, which include the aniline dyes as well as drugs (such as antipyrine, acentanilid, phenacetin), and such poisons as potassium chlorate, chloramine and carbon monoxide. Since the mortality rate among such cases is very high, he thinks exchange transfusion is of extreme value, since it is fairly easy to perform and is practically free of risks.

Petit and Herbaut[3] reported a case of accidental ingestion of phenylbutazone tablets in which exchange tranfusion played part in the successful outcome.

Boggs and Anrode[4] reported a case of boric acid poisoning treated by exchange transfusion. There is no antidote for boric acid, and recovery depends on the ability of the kidneys to excrete the substance before tissue concentrations are such as to cause irreversible or lethal changes. Goldbloom and Goldbloom,[5] in reviewing 109 cases, found the mortality rate to be 70.2% in infants under one year of age when all routes of administration were considered. Both McNally and Rust,[6] and Young, Smith, and MacIntosh[7] report fatalities in 12 infants after a single feeding of boric acid,—in the first series the infants received between 3 and 5 Gm. of boric acid, and in the second series 1 and 2 Gm. In discussing their case, Boggs and Anrode believe one must estimate how much boric acid is removed from the infant by exchange transfusion, and since there is no antidote or specific therapy for boric acid poisoning, exchange transfusion, using a quantity of blood approximately four times the blood volume, is necessary. In view of the high mortality rate reported in similar cases they believe this procedure materially contributed to the recovery of their patient.

In discussing exchange transfusion Bruton[8] points out it has become a common procedure in most hospitals in the United States. He indicates that no one can estimate the amount of poison ingested, and since dialysis of any type is technically difficult and not available to most hospitals, exchange transfusion is effective, and may be quickly provided in selected cases of poisoning. He illustrates this by results in three cases: one of a 2 year old child who swallowed several capsules of Nembutal; the second a 3 year old girl who had ingested several tablets of Pyridium; and the third a 28 month old girl who had ingested a large quantity of aspirin. Bruton believes that for the ex-

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change to be most effective it should be started at the earliest time possible after evaluation of the patient and the circumstances surrounding the poisoning permits. However, exchange transfusion will be indicated in only a small per cent of patients admitted for acute poisoning.

Katz and Carver[9] reported on the case of a 19 month old child who had ingested 9 isoniazid tablets of 100 mg. each, and although most of the effects from isoniazid therapy are referred to the nervous system, this patient fully recovered with no residue. Radebaugh and Emery[10] reported the case of a 2 year old boy admitted to the hospital with history of vomiting for 40 hours and rapid breathing for 38 hours. At first it was thought that this child might have diabetic acidosis, though a catheterized specimen of urine showed a pH 4, acetone 4-plus and only a trace of sugar. This diagnosis was abandoned when it was found that his blood showed a salicylate level of 39.8 per 100 ml. Though he was considered to be in extremis an exchange transfusion produced successful recovery.

Done and Otterness,[11] after commenting upon the toxicity of methyl salicylate and death resulting from ingesting as little as 4 to 7 ml., report the case of a 2 year old child who was seen 25 minutes following the ingestion of approximately 30 ml. of oil of wintergreen, and was treated by exchange transfusion. Data concerning the removal of salicylate during exchange transfusion and its influence on the concentration of salicylate in the plasma were studied. During the first two hours of exchange transfusion a total of 415 mg. of salicylate was eliminated (305 mg. by the procedure itself, and 110 mg. through urinary excretion), resulting in a reduction in salicylate level of the plasma from 84 to 52 mg. per 100 ml. During the subsequent one and a half hours, despite the elimination of an additional 391 mg. of salicylate, no further reduction in plasma salicylate level occurred. This finding most likely reflects an equilibrium between salicylate in the blood and that in tissues and/or the extravascular fluid compartment. It can be surmised that the degree of "tissue saturation" with salicylate would depend, at least in part, on the interval of time that had elapsed after the ingestion of salicylate. Thus, in cases where the necessity for the artificial removal of salicylate seems imminent, it would seem that the sooner such a procedure is accomplished, the better the result one might anticipate. The total amount of salicylate eliminated during the entire exchange transfusion was 806 mg. (542 mg. by removal of blood and 264 mg. through urinary excretion). This represents a rate of elimination of salicylate of 226 mg. per hour (152 mg. attributable to the procedure itself, and 74 mg. to urinary excretion). Prior to exchange transfusion the average rate of salicylate excretion was 75 mg. per hour. Thus, there was a threefold increase in the rate of salicylate elimination.

In reviewing the literature on exchange transfusion, Tauscher and Polich[12] pointed to exchange transfusion as the accepted form of treatment for hyperbilirubinemia in hemolytic disease of the newborn since Mollison and Walker's[13] studies published in 1952, and its more recent use for the removal of toxic substances from the blood and tissues in acute poisonings of children. Derot and associates[14] reported the use of exchange transfusion as an adjunct to the successful treatment of sodium chlorate poisoning, and DeCortis and coworkers[15] suggested its use in carbon monoxide poisoning. Tauscher and Polich[12] reported a case of a 4 year old child admitted to the hospital with tentative diagnosis of encephalitis. Following ingestion of candy, carbonated beverage and ice cream she became nauseated and vomited one time; on the next morning she had anorexia and was nauseated, so she was given an enema and shortly afterward became listless and lethargic. On admission this child was acutely ill and comatose. The odor of pine oil was detected on the patient's breath. Upon further questioning of the mother it was found that she had used 2 tablespoonfuls of a commercial disinfectant, Germtrol, in the enema that the child had never expelled. Though the patient appeared to be in a terminal state exchange transfusion was decided upon. The outcome in this case was successful. The toxic ingredient in Germtrol is pine oil, which comprises 49% of its composition. Though there have been no reports of dialysis or exchange transfusion used for this type of poisoning, the authors believed it justified because of the failure of supportive therapy. Since this patient responded so well, and because of the relationship of pine oil to turpentine in clinical symptoms, they believe it would be advis-

able to use exchange transfusion in severe cases of poisoning with turpentine or other poisoning with hydrocarbons. Because the widespread use of the artificial kidney is still limited, exchange transfusion should be the procedure of choice.

Case Report

We would like to present the following case. The patient was a 2 year old white boy who ingested an undetermined amount (approximately 15 cc.) of a liniment that contained oil of wintergreen. Two hours later he became irritable, crying and vomiting three times. The vomitus had an odor of oil of wintergreen. He was seen by the attending physician and given phenobarbital 0.03 Gm. for the excitement that was present, and then was admitted to the hospital.

Physical Examination. On admission, 3½ hours after the ingestion of the poison, T. was 100°, P. 160, and R. 36, and B.P. 110/10. He was well developed and well nourished, and was irritable when aroused. Hydration and color were satisfactory. There was no odor noted on the breath. Respirations were rapid and deep; the lung fields were clear. The heart had a normal sinus rhythm; no murmurs were noted. On inspection the abdomen was flat, and no organs or masses were palpable.

Course in Hospital. On admission a gastric lavage was performed. The stomach contents did not have the odor of oil of wintergreen. Blood was drawn on admission and the following found: CO_2 18.5, chlorides 111.1, potassium 5, and sodium 146 mEq./L. The blood salicylate level was 164 mg. per 100 ml. WBC. was 22,700 with a differential of 1 eosinophil, 2 bands, 71 segs., 22 lymphs., and 4 monos. The Hgb. was 11.2 Gm. Urine showed a pH of 5.5 and a trace of acetone; there was no glycosuria at this time.

The child received oxygen intermittently by mask. The general condition of the child continued to become worse and he became more drowsy shortly after admission. A "cut-down" was performed, and using Pediatric Solution 1:5, approximately 800 cc. were given in the first two hours. Four hours after admission, there being no improvement, a blood exchange transfusion was performed because of the possibility of death following the ingestion of much greater than the lethal dose of this drug. A total of 1,500 ml. of O+ blood was given through the "cut-down" on the left greater saphenous vein, and 1,250 ml. of blood removed from a "cut-down" on the right greater saphenous vein. The patient received, during the exchange, 20 ml. of 10% calcium gluconate in 250 ml. of 5% D/W. He tolerated the procedure well, which took approximately 1¾ hours. Following exchange the level of blood salicylate was 87 mg. per 100 ml.; the CO_2 was 18.2, potassium 3.4, chloride 94.5 and sodium 147 mEq./L.

The child was placed in an oxygen tent with vapor. Six hours after admission the T. was 98.5, P. 100, R. 24, and B.P. 88/50. Respirations were noisy and somewhat labored. Pediatric Solution was allowed to drop at 10 to 12 drops per minute with 100 mg. of Achromycin added in the first 550 ml. bottle after the exchange. The following morning the blood showed a CO_2 of 17.1, sodium 135, chloride 103.6, potassium 2.7, and blood salicylate of 77.6 mEq./L. The patient's general condition was very much improved with respirations 29 per minute and pulse 120. The child's voice was hoarse, and bilateral rhonchus was present. The Pediatric 1:5 fluids were continued with 20 mEq./L. of potassium added. The child was voiding well and by the end of the second day of hospitalization he was taking fluids by mouth. On the third day of hospitalization the blood chemical studies showed a CO_2 of 17.9, chlorides, 112.7, potassium 4, and sodium 144 mEq./L. The chest was clear and the vapor jet therapy was discontinued. Since the oral intake was adequate the "cut-down" treatment was stopped. Mild thrombophlebitis was present due to the "cut-down." The child remained afebrile and the area of phlebitis decreased. The patient was discharged on the 5th day of hospitalization in good condition.

Discussion

The ingestion of toxic drugs by young children is becoming a more and more serious condition. Certain drugs are usually fatal and the lethal dose may be very small. Many patients will get well if the electrolyte balance can be maintained and the toxic drug excreted, but certainly a good number of these children cannot get well under ordinary conditions.

The use of the extracorporeal dialysis, while of great value, cannot be successfully used because it is not available in the average community, and one must be acquainted with its use to accomplish the results wanted. The use of exchange transfusion, however, is now universally used and, with little effort, can become available in almost any part of this country.

In summarizing the indications for immediate dialysis or exchange transfusions, particularly in salicylate intoxication, we would like to quote Spritz and associates.[16] "They feel that coma, hyperpyrexia, seizure, delirium and other manifestations of severe central nervous system involvement need be corrected. They feel that these signs, and also metabolic abnormalities, cannot be easily managed by replacement therapy."

We agree with them that the concentration of the salicylate in the blood cannot be used as a criterion for prognosis. One must use his judgment in following the patient's course rather than the drug level and blood concentration. We also believe that the method of extracorporeal hemodialysis may be of greater value, but the simplicity of the exchange

transfusion lends itself to the choice of treatment in poisoning in children. We believe that in the hands of an experienced operator one can observe changes in the blood volume and the effects of transfusion on the patient as it is done in the use of this method in the correction of incompatibilities in the newborn.

We feel that in our case, after the ingestion of such a large amount of salicylate, this procedure was lifesaving.

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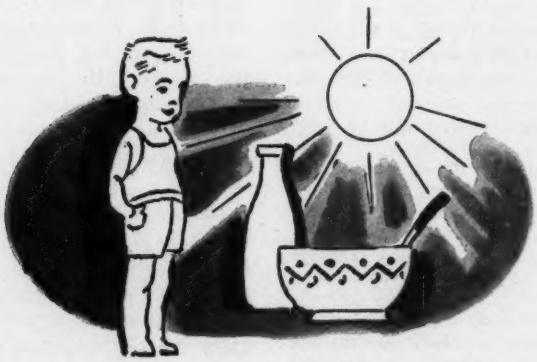

Stool Examinations in General Practice*

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FOR STOOL EXAMINATIONS, the most helpful knowledge is to know when to examine a patient's stool for parasites.

In the literature, it is said that an eosinophilia of 4% or more in a routine blood count is not a conclusive indication for examination of stools. However, if the patient is anemic, has an eosinophilia, a "pasty" color, and is without a history of allergy, a routine stool examination seems indicated. Thus, one will find a high evidence of positive results. About 10% of the patients in the present series with positive stool examinations had no eosinophilia; from 15 to 20% who had an eosinophilia did not have intestinal parasites. Though intestinal infestation with parasites is world-wide, our section of the country also shows a high evidence of infestation. Therefore I will confine my statistics to this area. Surveys have been made in many of the southern states, and checked years later to determine the amount of infestation present.

Though no figures are available for Florida for a complete survey, I have some figures for review on examinations made at the State Board of Health Laboratories. From 1954 through 1958, 666,688 stool examinations were done; 20.4% were positive for one or more parasites. This does not include the use of special containers nor special fixation for *Entamoeba histolytica.*

The common type worms found were: hookworms in 12%, and roundworms in 4.4%, others being found in proportion. Thus, one of every 5 stools examined was found positive for intestinal parasites.

The Rockefeller Sanitary Commission Survey in Alabama (1911 to 1914) revealed an average hookworm rate of 53.6 per cent. This was checked from 1931 to 1934; it was found there had been a marked drop in hookworm infestation. The rate was reduced to only 15.3%, and a similar reduction was shown for *Ascaris* and *Trichuris.* Another Survey was done in 1951 and 1952, in the heavily infected areas, with still further reduction.

Studies in Kentucky in 1954 revealed the following: *Entamoeba histolytica,* 3.3%; *Ascaris,* 27%; *Trichuris,* 25%; *Giardia lamblia,* 9.5 per cent. Tennessee studies in school children 1949 to 1950 for intestinal parasites were positive in 35%, as: hookworm 19.6%, roundworm 6.1%, pinworms 1.4%, *Amoeba histolytica* 3.06%, *Giardia lamblia* 9.6 per cent.

The history of amebiasis, an inclusive term embracing all phases of parasitization of man, by *Entamoeba histolytica,* is replete with discrepancies. Lambl, in 1859, erroneously identified *Trichomonas hominis* as an ameba. Kartulis, in his diagnostic enthusiasm, reported in 1886, the presence of amebae in each of 150 dysentery patients who were examined! Many believe these two early workers have present-day adherents.

The national incident of amebiasis has been estimated at an alarmingly high figure on the basis of repetition of historic, but authoritative surveys, specialized studies on a restricted population segment, and inaccurate estimates from unqualified observers. Standardization of technical procedures and higher requirements for qualification of technicians may minimize these discrepancies. The national frequency may approximate 4 per cent. Replies to questionnaires sent to the membership of the American Gastroenterological Association, and the American Association of Medical Clinics, as well as to parasitologists of medical schools of the United States, coupled with publications of 1933, to 1953, yield an estimate of 3.7 per cent. This national average must be adjusted for various specific localities. For instance, in the southern coastal states, the incidence is 7.5%, whereas the four states bordering Mexico show an average frequency of 5.8 per cent.

Historically, hookworm infestation existed in the early Dynasty of Egyptian civilization. The disease was described by many writers in Italy, Arabia, and Brazil long before the cause

*Chairman's Address, read before the Section on General Practice, Southern Medical Association, Fifty-Third Annual Meeting, Atlanta, Ga., November 16-19, 1959.

was discovered in 1838. In 1886, hookworms were found in patients dying with tropical anemia, and in 1878, it was discovered that infected individuals could be detected by the presence of eggs in the stool. Hookworm diseases were recognized in the United States as early as 1845, but the type of worm found here, *Necator americanus,* was not described until 1902.

The pathogenicity of parasites for man is still questionable in many instances, but of the protozoa, *E. histolytica,* and *Balantidium coli* are recognized pathogens.

The flagellates are questionable. The protozoans are important both from the diagnostic as well as from the environmental aspects. The methods of transmission are known to be of two types. The *direct* (pinworms): eggs are deposited in the stool and around the anal orifice, and are passed to the mouth, ingested and develop into worms in the intestinal tract. The *indirect* (roundworms): the eggs pass out in the stools to contaminate the soil, where they develop to the infectious stage, are ingested (roundworms) by food, or penetrate the skin (hookworms), to pass through the bloodstream to the intestinal tract.

Report of Stool Examination

This accumulation of 1,193 stool examinations was started in 1938, and completed in 1958, excluding five years during service in World War II. Most of the patients were asymptomatic.

Table 1 indicated the classification of intestinal parasites, both protozoa, and nematodes, and is self-explanatory. Table 2 shows the incidence among the white and colored races. The 766 patients with positive stools are divided by sex and race. The per cent of Negroes is small, since my practice includes but few of this race. Though not shown in the table, my records reveal 128 combinations of infestation, or 16.7% multiple infestations. It should be noted that *Trichuris* is not recorded, since the method of examination for this parasite does not depend on the stool examination.

Table 3 provides the incidence in age groups.

This is interesting since, contrary to our general belief, we find the highest incidence, 35.1%, in the age groups of 25 to 50 years. I did not suspect this until the records revealed it. The 7 to 15 year group is second, with an incidence of 31.3%, and the 15 to 25 year group is third with 19.4 per cent. Under 7 years of age the incidence is only 9%; over 50 years it is 5.2 per cent. This indicates the necessity of stool examinations. Some of us have the habit of treating children routinely for worms, without stool examinations; this chart shows how bad this guessing really is.

Table 4 shows the incidence of various pathogens in this series. *G. lamblia* are shown

TABLE 1

Intestinal Parasites	Protozoa	Pseudopods	*E. histolytica*
			E. coli
			Dientamoeba fragilis
			Endolimax nana
			Iodamoeba buetschlii
		Flagella	*Chilomastix mesnili*
			Giardia lamblia
		Ciliated	*Balantidium coli*
	Nematodes		*Enterobius vermicularis*
		Hookworms	*Necator americanus*
			Strongyloides stercoralis
			Trichuris trichiura
			Ascaris lumbricoides

TABLE 2

INCIDENCE

	Total	*Per Cent*	*Positive*	*Per Cent*	*Negative*	*Per Cent*
White female	481	88%	295		186	
White male	578		359		219	
Colored female	54	12%	37		17	
Colored male	80		75		5	
	1,193		766	64.2%	427	35.8%

TABLE 3

INCIDENCE IN WHITE AND COLORED MALES AND FEMALES IN AGE GROUPS

Positive Stools	*Under 7*	*7 to 15*	*15 to 25*	*25 to 50*	*Over 50*
Colored males	11	23	27	14	0
Colored females	3	8	17	9	0
White males	31	107	44	140	37
White females	23	102	61	106	3
Totals	68	240	149	269	40
Percentage	9%	31.3%	19.4%	35.1%	5.2%

TABLE 4

INCIDENCE OF VARIOUS PATHOGENS

	Colored		White	
	Male	Female	Male	Female
Negatives	5	17	219	186
E. histolytica	6	16	206	192
Hookworms	3	4	60	50
Roundworms	64	6	29	20
G. lamblia	2	11	59	32
Tapeworms	0	0	5	1

since they are questionable, though are found most commonly with other parasites, particularly *Entamoeba histolytica*.

Method

Our method of stool collection consists of a liquid diet the day before the patient comes for examination. At 7:30 a.m., on the morning of collection, a substantial dose of a saline cathartic is taken. The patient reports at my office by 8:30 a.m. where he is given three containers, marked 1, 2, and 3, and instructions on how to collect each sample. These are examined as soon as possible after collection, and are kept in the incubator at body temperature until examination is finished. The *Entamoeba histolytica* must be found moving, and must have a red blood cell in the cytoplasm for diagnosis, preventing confusion with the nonpathogenic type. Thereby, the laboratory technician does not make erroneous diagnoses. I sometimes check positive specimens when they are found. Concentrated salt mounts are made routinely, thereby revealing positive findings that may be missed otherwise. Most technicians can identify the ova of the many worm infestations.

Most of the patients in this survey were nonsymptomatic. This is the purpose of the paper, not to use patients having symptoms, but to use those one would normally not check the stools for parasites.

Treatment

There are many variations of treatment for parasites. The nematodes are not too difficult for treatment. Hexylresorcinol is satisfactory and very effective. I usually order two courses of the drug, one week apart.

Many new drugs are described each year for the treatment of protozoa (amebae). Most are effective in a great percentage of cases, but the small percentage not affected by therapy are the real problems. One reads that ten day treatments are sufficient, but in my experience this has not been true. I use two ten day treatments with Corbosome, with five day rest periods, and then follow this by two ten day treatments with Diadaquin, and still have about 5% of cases in which the stools remain positive. Emetine will help this group.

Some thought has to be given to reinfections. Five of these 10 cases, I am sure, were reinfected from surface water, but in the other 5 this was ruled out.

The one thought I would like to leave with you, is not to desert these people, but keep checking the stools, and trying by other drugs, to get the stools negative.

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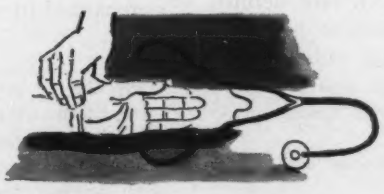

Deanol in the Treatment of Angina Pectoris: A Preliminary Report

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SINCE THE INTRODUCTION of nitroglycerin over 80 years ago in the treatment of angina pectoris,[1] no single drug or combination of drugs has surpassed it for immediate relief of pain. Development of long acting nitrites has provided some degree of effective prophylaxis against anginal attacks.

A prophylactic approach to the treatment of angina pectoris based upon anatomic and physiologic principles has been described by Chevalier and Simon.[2] Considerable attention has been directed to one of these, elevation of the cortical threshold of pain. Amino oxidase inhibitors are effective in a significant percentage of patients, but have marked limitations because of production of serious side actions. In our search for a prophylactic antianginal agent, which would be nontoxic and effective, a clinical trial with deanol+ was undertaken. Deanol* (dimethylaminoethanol p-acetamidobenzoate), a precursor of acetylcholine, has been used chiefly as a cerebral stimulant.[3] Extensive pharmacologic and clinical studies have demonstrated a wide therapeutic margin of safety, with no serious toxic effects within a fairly wide range of dosage.[4-6] Deanol activates the MDAS (mesodiencephalic activating system), with a less persistent and intense response than certain other stimulants.[7] The present report presents our experiences to date with this new therapeutic approach.

Methods and Materials

Nineteen patients with persistent angina pectoris were selected. Many had previously had myocardial infarction. All had definite clinical evidence of arteriosclerotic heart disease. Three additional patients with less definite anginal syndromes were also included. Deanol was initially given in a dosage of 25 mg. 3 times daily; the dose was later adjusted to the patients' requirements. The patients selected were those in whom nitroglycerin requirements could be estimated fairly accurately on the basis of weekly consumption. All of the patients either had angina of effort, decubitus, or angina associated with emotional factors.

A double-blind study was not done since this alone can not control the variables present in the anginal syndrome.[8] The average daily total number of nitroglycerin tablets was recorded as our reference criteria for the periods both before and after treatment with deanol.

The response was graded as follows: (1) *excellent*—patient had complete relief of pain, with increasing effort tolerance, requiring only very occasional nitroglycerin, or none at all; (2) *good*—patients in whom the nitroglycerin requirement was reduced at least 50% or more, and who obtained a clinically satisfactory response to pain; and (3) *no response*—patients in whom there was no decrease in the nitroglycerin requirement and who obtained no significant relief from pain.

The period of observation ranged from a minimum of 3 weeks to a maximum of 7 months. The drug was discontinued in patients who failed to respond within 2 weeks. All patients were hospitalized for periodic evaluation, and serial laboratory data were obtained. These included hemograms, electrocardiograms, blood urea nitrogen, and electrolyte determinations. Other ancillary studies were included as indicated. The results are summarized in table 1.

Results

Of the 19 patients with angina, 60% obtained some therapeutic response. Four had an excellent response, 8 a good response and 7 obtained no significant response. One patient, with angina decubitis, and who was in the nonresponsive group developed fatal myocar-

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*Supplied as Deaner by Riker Laboratories, Inc., Northridge, Calif.

TABLE 1

Patient (Age and Race)	*Diagnosis*	*Weekly Nitroglycerin Requirement*	*Response*	*Remarks*
(1) J.W. 40—W	ASHD Myocardial infarction 8 mos. before adm.	10-15	Excellent response within 3 das.	
(2) J.M.R. 44—W	ASHD Myocardial infarction 5 yrs. prior to adm.; duodenal ulcer; diabetes mellitus	8-12	Good	
(3) C.M. 51—W	Psychogenic C-V-reaction	18-20	Excellent	Marked cardiac consciousness; normal resting EKG.; single and double Master's two-step test normal
(4) A.F.E. 64—W	ASHD Myocardial infarction 4 mos. before adm.	14-20	Excellent	No response to long acting nitrates; free of pain on 1 tab. deanol daily
(5) R.M. 40—W	HCVD ASHD Myocardial infarction 3 mos. before adm.	8-14	Good	
(6) N.J.S. 37—W	R.H.D. with mitral stenosis	8-12	Good	Frequent anginal chest pain; previous unsuccessful mitral commissurotomy; insomnia relieved by reduction of deanol to 25 mg. b.i.d.
(7) C.N. 41—W	ASHD Myocardial infarction 9 mos. prior to adm.; idiopathic hypercholesterolemia	10-16	Good	
(8) T.R.B. 50—W	ASHD Myocardial infarction 2 mos. prior to adm.; chronic alcoholism; duodenal ulcer	20-35	None	Marked emotional lability
(9) L.W.H. 40—W	ASHD Myocardial infarction 4 mos. prior to adm.; paroxysmal tachycardia	15-20	Excellent	Died 6 mos. later; recurrent acute myocardial infarction
(10) H.F.Q. 49—W	ASHD Recurrent myocardial infarction; 2 yrs. before adm.	15-20	Good	Marked emotional overlay
(11) H.B. 52—W	HCVD Myocardial infarction 2 yrs. before adm.	16-22	None	Died with recurrent myocardial infarction 8 mos. after onset of angina
(12) J.T.B. 42—W	Cervical neuropathy secondary to trauma	7-12	Good	Anterior angina-like chest pain; was told he had heart disease; resting EKG. normal; single and double master, two-step test normal; marked emotional overlay
(13) H.J. 41—W	ASHD Myocardial infarction 3 mos. before adm.	10-12	Good	
(14) O.C. 66—W	ASHD Recurrent myocardial infarction; abdominal angina	8-10	None	Responded to long acting coronary vasodilators; marked psychogenic component
(15) C.V. 54—W	ASHD Old myocardial infarction	12-16	None	Developed subendocardial infarct.
(16) F.S. 50—W	HCVD ASHD Myocardial infarction 2 yrs. before adm.; duodenal ulcer	10-20	Good	Residual complaints suggested duodenal ulcer; responded to anti-ulcer regimen following relief of chest pain
(17) T.V.I. 62—W	ASHD Old myocardial infarct	12-15	Good	Emotionally labile
(18) E.D. 64—W	ASHD Old myocardial infarct	10-15	Good	
(19) C.P. 62—W	ASHD Duodenal ulcer; psychoneurosis	15-20	None	Marked anxiety and concomitant G.I. complaints; evaluation of therapy impossible
(20) H.B. 46—W	ASHD 3 previous myocardial infarctions	6-8	None	Pain subsided spontaneously while on deanol.
(21) D.M. 47—W	ASHD 2 previous myocardial infarctions	20-24	Excellent	
(22) A.M.	ASHD Recent myocardial infarction	10-12	None	Pain subsided spontaneously while on nonspecific therapy

ASHD—Arteriosclerotic heart disease.
HCVD—Hypertensive cardiovascular disease.
R.H.D.—Rheumatic heart disease.

dial infarction while on the medication. Another patient developed a nonfatal subendocardial infarction which was thought to be present previous to institution of the medication. Three additional patients with angina-like syndromes had good therapeutic results.

Discussion

An evaluation of any drug which is based primarily on symptomatic response must be attended with utmost caution. The enthusiasm of the observer can easily becloud interpretation of results. Of even more significance are the vagaries of the anginal syndrome. That spontaneous periods of freedom from discomfort may occur, especially under hospital supervision, is well known. We know of no tests or devices that can measure the anginal episode or its response to treatment. Probably the most dependable aid is the therapeutic response to nitroglycerin. However, many anginal-like episodes also respond to this drug. We utilized the number of nitroglycerin tablets, on an average weekly basis, as an indirect measure of the effectiveness of deanol.

It has been stated that agents which block pain at the cerebral level are not without danger. Towers and Wood have actually advised the concomitant use of anticoagulants if the "warning pain" is abolished. We believe this requires further study. Of interest in this regard is patient No. 11 in whom deanol was ineffective; this patient noted persistent pain and developed subsequent fatal infarction. Patient No. 15 also failed to respond to deanol and developed electrocardiographic changes consistent with a subendocardial infarction. It would appear, therefore, at least in these 2 instances, that the drug did not abolish the so-called "warning pain."

On the basis of pharmacologic studies, it would seem that deanol potentiates block in the central nervous system to a lesser degree than do other similar agents, and does not interfere with the "alarm mechanism." Master[10] noted no change in the electrocardiogram on "two-step tests" with iproniazid. One of his patients developed an episode of coronary insufficiency, and another died following an acute coronary occlusion while on the drug. Thus it seems that these drugs do not disrupt entirely the "cardiac alarm system" as has been postulated.[11] Suppression of the anginal pain to the subthreshold level, therefore, does not occur as frequently as predicted, and probably less so. Obviously, further evaluation will be needed to clarify this somewhat controversial point.

Side actions were seen in only one patient in this series. This patient complained of insomnia, which responded to reduction in dosage.

Conclusion

Nineteen patients with angina pectoris, and 3 patients with angina-like syndromes, were treated with deanol, in courses varying from 3 weeks to 7 months. Sixty per cent of the patients with angina had a therapeutic response —4 excellent, 8 good, 7 ineffective. It is believed that 2 myocardial infarctions in the group were in no way related to the medication. Three patients with angina-like pain are considered to have obtained good therapeutic responses.

On the basis of past clinical experience, no general toxic reactions were expected; none were noted in either a general or specific (cardiovascular) sense. One patient developed insomnia which promptly responded to decrease in dosage.

Although no adequate control design may be introduced into this type of study, it is believed that deanol deserves further trial in the treatment of angina pectoris.

Addendum

Case 4. (A. F. E.) A 64 year old white man had an acute myocardial infarction in November 1958. In December 1958 and January 1959 the patient had recurrent anginal attacks, especially related to anxiety, tension and effort. His initial response to coronary vasodilators was unsatisfactory. From 14 to 20 nitroglycerin tablets were required each week. He was started on deanol in February 1959 and, within a period of 3 days, complete relief of pain with marked improvement in his effort tolerance was noted. He eventually returned to his business as a construction superintendent. He was seen at monthly intervals, during which time he had maintained himself on deanol, and he stated that with one 25 mg. tablet daily, he is able to prevent any recurrence of his attacks. If he misses one day, he experiences recurrent angina.

Case 11. (H. B.) A 52 year old white man had a myocardial infarction approximately one year before admission. Because of recurrent attacks of angina on effort, and angina decubitis, which persisted for approximately 8 to 10 months preceding admission, the patient was started on deanol. His nitroglycerin requirement had varied from 16 to 20 tablets weekly. The patient was again hospitalized for sustained hypertension. He continued to have anginal pain and subsequently developed a second myocardial infarction; he

died in his sleep approximately 8 days after onset. Deanol was ineffective in relieving this patient's pain.

Case 15. (C. V.) A 54 year old white man with a history of an old myocardial infarction was hospitalized because of persistent anginal pain. On admission, and during the first several days of hospitalization, EKG. studies revealed no evidence of recent changes. He required approximately 12 to 16 nitroglycerin tablets weekly. He was started on deanol, and approximately one week later electrocardiographic changes consistent with recent subendocardial infarction appeared. His response to deanol was ineffective and, following discontinuation of all medication, he was symptom-free although his effort tolerance was moderately limited.

Case 8. (T. R. B.) A 50 year old white man who had a long history of alcoholism, duodenal ulcer and emotional instability was admitted because of chest pain. Studies revealed a recent myocardial infarction. The anginal type chest pain persisted for some 3 or 4 months after infarction, with no further deterioration of his electrocardiogram. His response to coronary vasodilators was poor, and he required from 20 to 35 nitroglycerin tablets weekly for relief of pain. After approximately one week on deanol his pain was unimproved. Incidentally, with iproniazide the frequency of his attacks continued; he also developed lightheadedness and severe headache necessitating discontinuation of this drug.

Case 6. (N. J. S.) A 37 year old white man, with a history of chest pain, was known to have rheumatic heart disease with mitral stenosis and insufficiency. He had had a technically unsatisfactory commissurotomy approximately 3 years previously. He obtained relief with nitroglycerin and used approximately 8 to 12 tablets weekly. On deanol 25 mg. 3 times daily he obtained marked relief of pain and required no nitroglycerin. Because of insomnia, deanol was reduced to 25 mg. twice daily, which abolished his insomnia. He had no further anginal type pain.

Case 3. (C. G.) A 51 year old white man had been hospitalized on several occasions because of chest pain. Over a period of approximately 6 to 8 months the patient had been taking from 18 to 20 nitroglycerin tablets weekly for relief of chest pain. Pain was related mostly to tension and nervousness, but at no time was his EKG. abnormal. Single and double two-step Master's exercise tolerance tests were entirely within normal limits. On deanol 25 mg. 3 times daily the patient had no further complaints of pain.

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Preliminary Program

54th Annual Session

Will Appear In

October Issue

EDITORIALS

MILK INTOLERANCE—CLINICAL EVALUATION

For several decades an occasional article has appeared in the medical literature on a symptom-complex in which milk has been accused as the cause of the manifestations, either in a direct way, as atopic eczema, or in a combination of anorexia, pallor, ease of fatigue and the like.

Let me hasten to say that I approve the good nutritional value of milk and its place in the American diet. At the same time it has limitations and contraindications in usage. The universal axiom of a "quart of milk per day for every child" is a very misleading statement. Nor is the use of milk fat without critical analysis.

The expression of "allergy to milk" is frequently frowned upon by our allergy-minded colleagues. Even though skin and transfer reactions cannot often be obtained, clinical evaluation can be the "proof of the pudding."

The answer to reactions in other organs of the body (aside from atopic eczema) may easily lie in the area of biochemistry or the enzyme system. The mucosa of the gastrointestinal system has been shown to evidence a local reaction when milk has been applied directly. However, when causing other symptomatology, one of its amino acid components is the logical supposition.

Beside the eczema (easily recognizable) and the recently accepted causation for a percentage of "colic," other less obvious syndromes can be demonstrated. Among these are certain symptoms in older children of school age. Among these may be repeated: pastel complexion, ease of fatigue, irritability, poor school attitudes, constipation, low-grade fevers, convulsive seizures resembling epilepsy (abnormal EEG.), and other similar reactions. Reduction of, or total abstinence from milk in the diet is essential to improvement. No animal drinks milk after cutting teeth except those domesticated ones—including man!

The above observations are purely clinical and set down for use on a trial basis where failure is a harmless result.

PRESTON A. MCLENDON, M.D.

THE EFFECT OF RADIANT ENERGY

This issue includes a paper outlining the "Adverse Effects of Sunlight on the Skin."[1] It reminds one that radiant energy is a most potent physicochemical force, a matter commonly forgotten by the physician and one about which the layman is woefully ignorant. Possibly it is timely to recall some of these facts, especially in these days of the cult of sun worship and prolonged and repeated exposure of much of the body surface to a potent physical agent.

Much has been learned over the years of the effects of sunlight biologically. However, most of this information has been published, not in the clinical literature, but rather in journals and publications dealing with the basic sciences and biology. Only here and there has some of this subject matter cropped up in the journals read by the average physician. (The literature in the field of dermatology is an exception.) In the past couple of years much more has been written for the clinical journals because of the interest in the psoralens. These are the substances which when taken orally increase the pigmentation and thickening of the epidermis. Actually, a Symposium was held on the subject and radiant energy two years ago, the papers being published in early 1959.[2]

In the Symposium much of the information which had been accumulating over the years was brought together for the clinician. The papers on "Photokinetics," on "Solar Radiation and the Environment" and on "The Physiological Effects of Sunlight" are of particular interest and revealing to the casual reader, as well as those dealing with the melanocyte and the physiologic factors influencing its function.

This group of papers is informative as well as interesting, and thus one may learn of the chemical and cytologic aspects of melanin and its formation. So too, one learns of substances which catalyze photochemical reactions and

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the transfer of light energy to carotenoid pigment. It seems that proteins may transfer light energy to conjugated dyes and *vice versa*. The wave length of ultraviolet light appears of importance insofar as effect is concerned. Thus, the erythema from certain wave lengths occurs because of the absorption of the light by tyrosine and tryptophane. The author (Daniels) writing on photokinetics for the Symposium emphasizes the prolonged effect of ultraviolet light, commonly unappreciated, saying "the vascular effects of a single moderately severe erythema producing exposure to ultraviolet light" lasts for four to fifteen months, and says of ultraviolet effects that we are "playing for keeps." The same author calls attention to the systemic effects of visible and ultraviolet light, such as the synthesis of vitamin D, the lowering of blood pressure, and hemorrhage from the lung in tuberculous patients. He calls attention to the several amino acids which act as photo-absorbers, as well as riboflavin, carotenoid pigment and melanin, which he names the "energy trap." He makes a significant statement, "Since relatively simple chemical reactions can be photosensitized by other chemicals, it is not surprising that biological systems can also be sensitized by various chemical substances. They may occur as abnormal metabolites, or be ingested as part of the diet, or may be given as drugs."

Another participant in the Symposium, Fowlks, whose topic was "The Mechanism of the Photodynamic Effect," discusses organic chemicals and biologic substances which may be photosensitized. He describes experimental studies with the effect of ultraviolet light on a protein-methylene blue complex which becomes photosensitized and thereby the effect of light upon the living cell may be evaluated. Damage to cells may be of varying degree. Drugs which have been shown to be photosensitizers at one time or another have been members of the phenothiazines, hydrocarbons, acridines, pyrimidines, and sulfonamides as well as quinine, diethyl stilbesterol, p-aminobenzoic acid and certain dyes. No doubt there are others.

Some of this information on photosensitization is of interest in regard to one item mentioned by Guin. Dermatologists have documented for more than half a century, their clinical observations of the ill effect of exposure to sunlight upon discoid lupus and the apparent conversion of this disease to systemic lupus, upon occasion. Furthermore, there are a number of reports in the literature of fulminant dermatomyositis following excessive exposure to sunlight. Though such a relationship may be difficult of proof, the apparent causal relationship is too dramatic at times to be idly dismissed. In these days of rapid advances in the studies on auto-sensitization and auto-immunization in the area of the so-called collagen diseases, one cannot help but speculate upon the effect of such a potent source of energy as sunlight in the production of damage to the living cell, either alone or with the abetment of a photosensitizing drug. If such were shown to be the case, speculation would then need to consider such damaged tissue as a source of auto-sensitization with potential resultant auto-immunization and the tissue reaction of disease.

In any event, speculation aside, sunlight should be recognized as a potent source of energy, and not be considered as only benignant by the Caucasian with his lightly pigmented skin.

ANNOUNCEMENT

Southern Society for Pediatric Research

The organizational and scientific meeting of the Southern Society for Pediatric Research will be held on October 29 and 30, 1960, at Vanderbilt University School of Medicine, Nashville, Tennessee. The aims of the new organization will be to promote basic and clinical research oriented toward the field of pediatrics in the southern region and to serve as a forum for those interested in pediatric education. The meeting will be open. Full and part-time members of Pediatric departments, practitioners and residents are invited. The membership will probably be elective. The deadline for submission of abstracts is October 1. Correspondence should be directed to Dr. Mildred Stahlman, Department of Pediatrics, Vanderbilt University School of Medicine, Nashville, Tennessee.

25 YEARS AGO

Treatment of Chronic Arthritis.*

"Sulphur in one form or another has been used as a therapeutic agent since ancient times. Only recently, however, has its value in the treatment of chronic arthritis been stressed. Among the earliest papers on the use of colloidal sulphur in the treatment of chronic arthritis are those of Comrie and Cawadias, both of whom report favorable results. . . . Wheeldon and Main report that, on the basis of 250 cases studied for 2 years, they were convinced that colloidal sulphur had a definite place in the therapeutics of arthritis. Woldenberg, employing colloidal sulphur in the treatment of 100 cases of atrophic arthritis, believed that it had a definite therapeutic value. . . .

"Sulphur metabolism does not come within the scope of this paper, but it should be pointed out that the cystin content of certain structures is considered a reliable guide to the sulphur metabolism of the body. Sullivan recently described a method for estimating the cystin content of the fingernails. Sullivan and Hess, in a study of the fingernails of 105 patients with arthritis, found the cystin content below normal in 65% of the cases. . . .

"Sullivan and Hess applied sulphur therapy to 6 patients with a subnormal cystin content of the fingernails and found that improvement was accompanied by a rise in the cystin content of the fingernails. Woldenberg also reported an increase in the cystin content of the fingernails after colloidal sulphur therapy.

"The present study was undertaken not only to evaluate the clinical results of colloidal sulphur therapy in chronic arthritis and its effect upon certain cellular and immune reactions of the blood, but to ascertain the effect of this form of therapy upon the cystin content of the fingernails and to determine whether the cystin determination is of value in selecting cases for sulphur therapy.

"The patients were selected on admission to the clinic without regard to type, severity, age, or duration of symptoms. The usual routine physical and laboratory examinations were made on all patients and the cystin content of the fingernails was estimated according to the method of Sullivan before treatment was instituted. . . .

"An aqueous solution of colloidal sulphur (Sulisocol) containing 10 mg. per cc. was administered intravenously or intramuscularly twice weekly. No other treatment was given. . . . Certain patients who received 10 mg. twice weekly for several weeks and who were not improved were definitely improved when the dose was increased to 20 or 30 mg. . . .

"Symptoms of fatigue, drowsiness, loss of appetite, headaches and increased pain, stiffness or swelling of the joints after the intravenous injection of colloidal sulphur indicate overdosage. . . .

"*Discussion.* The results of the present study, as well as the work of others, indicate that colloidal sulphur produces favorable clinical results in a significant percentage of patients with chronic arthritis. The exact mechanism by which improvement occurs is unknown, but enough information is available to enable one to speculate on its possible mode of action. Cawadias states that sulphur plays a very important part in the process of nutrition, particularly the nutrition of joints. Loeper and his associates have noted that the neutral sulphur of the joint cartilage in rheumatoid arthritis was reduced about one-third below normal while the total sulphur of the blood was increased. . . .

"Sullivan suggested that inasmuch as glutathione aids in detoxication, its deficiency, as a result of improper metabolism, might allow uncontrolled action of the noxious agent in the arthritic. He believed that the administration of sulphur in these cases prevented the normal body sulphur complexes from being diverted into other channels. A low blood glutathione and a low cystin content of the fingernails, therefore, might indicate that there is a deficient oxidation of sulphur in these cases and, as it apparently plays a part in oxidation and in the nutrition of the joints, this may be the underlying factor in sulphur therapy. . . .

"*Conclusions.* 1. Fairly large doses (10 to 30 mg. twice weekly) of colloidal sulphur, as a rule, are well tolerated.

2. With the larger doses, however, some patients developed symptoms of toxicity, such as fatigue, drowsiness, headache, increased pain, stiffness, or swelling of joints but they usually disappeared in 5 days and were never severe enough to cause anxiety.

3. When the cystin content of the fingernails was below normal, it was usually increased with sulphur therapy.

4. The percentage and degree of clinical improvement was greater in those patients with a subnormal cystin content than in those with a normal content.

5. The percentage and degree of clinical improvement was greater with sulphur than with placebo therapy.

6. Sedimentation rates and non-filament cell counts were reduced in those cases showing clinical improvement.

7. There was no significant effect upon the red and white blood cell counts.

8. We believe that colloidal sulphur is a valuable agent in the treatment of certain types of arthritis."

*Rawls, W. B., Gruskin, B. J., and Ressa, Anthony A.: The Value of Colloidal Sulphur in the Treatment of Chronic Arthritis, Am. J. M. Sc. 190:400, 1935.

SOUTHERN MEDICAL NEWS

State News Items

The St. Louis County Medical Society and Auxiliary will entertain Southern Medical Association at a cocktail party, dinner, and musical production on Sunday evening, October 30, just prior to the SMA meeting, October 31-November 3, 1960. The program will be at the Jefferson Hotel, beginning at 7:30 p.m. The cost of $8.00 per person includes cocktail party, dinner, show, tax, tip, souvenirs, etc. For reservations send a check stating the number of tickets and your name and address to Mrs. M. A. Diehr, 28 Dromara Road, Ladue 24, Missouri.

The American Urological Association offers annual awards totaling $1,000 for essays on the result of some clinical or laboratory research in urology. Competition is limited to urologists who have been graduated not more than ten years, and to hospital interns and residents doing research work in urology. For complete information write the Executive Secretary, William P. Didusch, 1120 North Charles Street, Baltimore, Maryland. Essays must be in his hands before Dec. 1, 1960.

The American Rhinologic Society will hold its sixth annual meeting at the Belmont Hotel, Chicago, October 8. Physicians are invited. There is no registration fee. For details, write Dr. Robert M. Hansen, Secretary, American Rhinologic Society, 1735 North Wheeler Avenue, Portland 17, Oregon.

The 17th Annual Meeting of the American Medical Writers' Association will be held at the Hotel Morrison, Chicago, November 18 and 19. The two day program will include papers on various phases of medical writing, the publishing and editing aspects of medical writing, and the technic of medical writing. A complete program may be obtained from Dr. Harold Swanberg, Secretary, AMWA, W. C. U. Building, Quincy, Illinois.

The next scheduled examination (Part 1, written) of the American Board of Obstetrics and Gynecology will be held on Jan. 13, 1961. For further information write Dr. Robert L. Faulkner, Executive Secretary and Treasurer, 2105 Adelbert Road, Cleveland 6, Ohio.

ALABAMA

Dr. Harry C. Shirkey, Associate Professor of Pediatrics at the University of Alabama Medical Center and Director of Children's Hospital, has been appointed to a 10 year term on the Committee for Revision of United States Pharmacopeia.

Dr. Don A. Olson, Assistant Professor of Speech Pathology at the University of Alabama Medical Center, is now an abstractor for the National Index on Deafness, Speech and Hearing.

Officers of the Alabama Dental Association include Dr. John Sullivan, Mobile, President; Dr. V. David Cooper, Birmingham, President-Elect; Dr. Frank Finney, Gadsden, Vice-President; Dr. Charles Crook, Montgomery, Secretary-Treasurer; and Dr. Paul Dawson, Scottsboro, Board Member.

Dr. William Coats, Chickasaw, is President of the Mobile Chapter of the Alabama Academy of General Practice; Dr. H. C. Mullins, Fairhope, is President-Elect; and Dr. Jack Coleman, Whistler, is Secretary-Treasurer.

Dr. William H. Robertson, Birmingham, is Assistant Secretary of the American Society for the Study of Sterility.

ARKANSAS

New members of various county medical societies include *Sebastian*: Drs. Mario A. Accinno, Donald J. McMinimy, and Stonie R. Cotton, all of Fort Smith; *Pulaski*: Drs. Harold D. Langston, Louis E. Tolbert, and Rodney Fitzgibbon, all of Little Rock; *Washington*: Drs. Harold E. Martin and Wade Burnside, Fayetteville; *Benton*: Dr. E. N. McCullum, Decatur; and Dr. Marvin L. Murphy, Siloam Springs; *Craighead-Poinsett*: Dr. Herman D. Alston, Jr., Jonesboro; *Crittenden*: Dr. H. G. Lanford, West Memphis; *Hempstead*: Dr. Lowell O. Harris, Hope; *Garland*: Dr. William J. Grippe, Hot Springs; *Pope-Yell*: Dr. James O. Pennington, Ola; and Dr. T. D. Robertson, Atkins; *Mississippi*: Dr. E. A. Shaneyfelt, Manila; *Union*: Drs. Grady E. Hill, Jr., and L. G. Fincher, Jr., El Dorado; *Jefferson*: Drs. Milton R. Wirthlin and Arlee E. Pollard, Pine Bluff.

New officers of the Brinkley Country Club are Dr. Pat Harrison, President, and Dr. J. P. Williams, Secretary-Treasurer, both of Brinkley.

Dr. Richard V. Ebert was re-elected President of the Pulaski County Tuberculosis Association and Dr. John E. Greutter, Jr., is Second Vice-President. Both are of Little Rock.

Dr. Wendell Garden is a new member of the staff at the Community Methodist Hospital in Paragould.

Dr. Oliver Wallace, Little Rock, has been re-elected President of the Carroll County Medical Society. Dr. W. A. Redman, Eureka Springs, was re-elected Secretary-Treasurer.

DISTRICT OF COLUMBIA

The Medical Staff of the Washington Hospital Center has elected: Dr. J. Spencer Dryden, President; Dr. Edgar W. Davis, First Vice-President; Dr. Thomas W. Mattingly, Second Vice-President; Dr. Francis L. Wenger, Secretary; Dr. Forrest V. Schumacher, Assistant Secretary; and Dr. Garnet W. Ault, Treasurer.

The Bureau of Mental Health of the D. C. Department of Public Health has announced that Drs. David A. Lanham and Irving L. Berman have joined the staff of the Legal Psychiatric Services Division. Dr. George Chornesky has been appointed Acting Chief of the Alcoholic Rehabilitation Division.

Dr. John W. McTigue has been elected to the Board of Trustees of the D. C. Society for the Prevention of Blindness.

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St. Louis Meeting Features Two "First Time" Symposia

Southern Medical Association is presenting two "first time" symposia at its Fifty-Fourth Annual Meeting in St. Louis, Missouri, October 31-November 3, 1960. On Monday morning, October 31, the Symposium on Cerebrovascular Disease will be featured and on Thursday morning, November 3, the Symposium on Medical Economics. These symposia should offer points of interest to all physicians. Nationally renowned guest speakers have been selected for each of the two programs.

The Symposia round out one of the most complete programs offered by any medical society. A total of 46 half-day sessions, covering every phase of medicine, have been prepared by 20 scientific sections.

Symposium on Cerebrovascular Disease

The subject of cerebrovascular disease embraces the fields of geriatrics, medicine, neurology, neurosurgery and vascular surgery, and is of wide general interest. As such it makes an excellent topic for a symposium. Dr. J. Garber Galbraith, Birmingham, Alabama, is Chairman of this Symposium. A native of Anniston, Alabama, Dr. Galbraith received his M.D. degree from St. Louis University School of Medicine. He interned at the Employee's Hospital of Tennessee Coal and Iron Corporation in Fairfield, Alabama, and later served a residency in neurosurgery at the Neurological Institute in New York City. Dr. Galbraith is neurosurgeon at Jefferson-Hillman Hospital, St. Vincent's Hospital, South Highlands Infirmary, Lloyd Noland Hospital and Children's Hospital, all in Birmingham. He is also Professor of Neurological Surgery at the Medical College of Alabama and has been certified by the American Board of Neurological Surgery. Dr. Galbraith is Councilor from Alabama to Southern Medical Association and a member of the Executive Committee of the Council.

The program outline is as follows. After introductory remarks by Dr. Galbraith, Dr. William S. Fields, Houston, Texas, will discuss "Current Concepts of Cerebral Vascular Insufficiency." Due to the advances made by arteriography, revision of some of the older clinical concepts of cerebrovascular disease is now necessary and desirable. Dr. Fields will review these advances and new concepts. A native of Baltimore, Maryland, Dr. Fields is an alumnus of Harvard Medical School. He is Chief Consultant in Neurology at the Veterans Administration Hospital, Neurologist-in-Chief of Jefferson Davis Hospital and senior neurologist at the Methodist Hospital, all in Houston. Dr. Fields is also Professor of Neurology at Baylor University School of Medicine.

Dr. Sheila Sheehan, Detroit, Michigan, has chosen the topic "Basilar and Carotid Artery Insufficiency Syndromes: Diagnosis and Medical Therapy." She will discuss the best medical means of maintaining adequate cerebral blood flow. Dr. Sheehan is a member of the Department of Neurology at Wayne State University College of Medicine.

The subject "Arteriography in Diagnosis of Occlusive Cerebrovascular Disease" will be explored by Dr. M. M. Keirns, Memphis, Tennessee. He will present the indications and contraindications for arteriography, along with slides demonstrating typical occlusive vascular lesions. Dr. Keirns received his M.D. degree from Harvard Medical School and interned at the Roosevelt Hospital in New York. He is currently on the staff of the Baptist Memorial Hospital in Memphis.

Dr. Francis Murphey, Memphis, Tennessee, is the next speaker scheduled. Under the

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Symposium on Cerebrovascular Disease

MODERATOR: *J. Garber Galbraith, M.D., Birmingham, Alabama*

Monday, October 31, 1960 *8:30 a.m.-12:00 noon*

8:30 INTRODUCTION
J. Garber Galbraith, M.D.

8:40 CURRENT CONCEPTS OF CEREBRAL VASCULAR INSUFFICIENCY
William S. Fields, M.D., Houston, Texas

9:05 BASILAR AND CAROTID ARTERY INSUFFICIENCY SYNDROMES: DIAGNOSIS AND MEDICAL THERAPY
Shelia Sheehan, M.D., Detroit, Michigan

9:30 ARTERIOGRAPHY IN DIAGNOSIS OF OCCLUSIVE CEREBROVASCULAR DISEASE
M. M. Keirns, M.D., Memphis, Tennessee

9:45 SURGICAL MANAGEMENT OF CAROTID INSUFFICIENCY
Francis Murphey, M.D., Memphis, Tennessee

INTERMISSION — VISIT EXHIBITS (30 minutes)

10:30 SURGICAL MANAGEMENT OF BASILAR AND CAROTID INSUFFICIENCY ASSOCIATED WITH OCCLUSIVE LESIONS OF AORTIC ARCH AND ITS BRANCHES
E. Stanley Crawford, M.D., Houston, Texas

10:45 RADIOLOGICAL DIAGNOSIS OF INTRACRANIAL ANEURYSMS AND VASCULAR ANOMALIES
M. M. Keirns, M.D., Memphis, Tennessee

10:55 MANAGEMENT OF INTRACRANIAL ANEURYSMS
Henry G. Schwartz, M.D., St. Louis, Missouri

11:25 HYPOTHERMIA IN MANAGEMENT OF CEREBROVASCULAR LESIONS
Hubert L. Rosomoff, M.D., Pittsburgh, Pennsylvania

11:45 PANEL — QUESTION AND ANSWER PERIOD

Symposium on Medical Economics

The physician, in addition to being a scientist, must also be a businessman for the practice of medicine has a business as well as a scientific side. The business side of medical practice requires the same control and planning as any business venture. Therefore, Southern Medical Association is presenting a Symposium on Medical Economics. This is designed to help the physician find the proper balance between the scientific and business aspects of medicine. Dr. Robert D. Moreton, Fort Worth, Texas, is Chairman of the Symposium. He was born in Brookhaven, Mississippi. After receiving his M.D. degree from the University of Tennessee, he interned at the Employee's Hospital of Tennessee Coal and Iron Corporation in Fairfield, Alabama. Dr. Moreton was a Fellow in radiology at the Mayo Foundation from 1940-42. He has been certified by the American Board of Radiology and is now Clinical Assistant Professor of Radiology at the University of Texas Southwestern Medical School in Dallas. He is Councilor from Texas to Southern Medical Association and is Chairman of the Executive Committee and of the Council.

Dr. Moreton has announced a program of four outstanding speakers. Two major sections will give practical advice and answers to problems the doctor has in the two environments where he spends most of his working time—the office and the hospital.

I. Doctor and His Office

John R. Sedgwick, Chicago, Consultant on individual and group medical practice for the Sears-Roebuck Foundation, will discuss "The Doctor as a Businessman." Mr. Sedgwick spent many years as the business manager of a medical clinic and is considered one of the best informed men today on the business side of medical practice. His topic is expected to include the problems of city and small town practice, such as how and where to get started, and how to organize and run an office or small clinic.

Woodrow Wirsig, New York, Editor of *Printer's Ink,* and former Editor of *Woman's Home Companion,* has chosen the topic "Yardstick for Action." He is well known in the publishing field. As a man familiar with the art of communication, he is well qualified to observe the human relations conduct of the physician as the doctor comes face to face with the patient. Mr. Wirsig has conducted personal interviews and compiled an image of the physician as seen by the public.

Horace Cotton, Oradell, New Jersey, a Senior Editor of *Medical Economics,* will speak on "The Business of Practicing Medicine." A frequent contributor to the editorial pages of *Medical Economics,* Mr. Cotton was for many years a consultant to physicians on the business management of practice. He will give specific "how to do it" suggestions on the daily details of office management. He is also expected to discuss the relation between the physician and his office assistant.

II. Doctor and the Hospital

Dr. Joseph F. Sadusk, Jr., Oakland, California, a member of the American Medical Association Committee on Medical Legal Problems, will discuss "Steps to Protect Yourself Against Professional Liability." A native of Baltimore, Maryland, Dr. Sadusk is a graduate of Johns Hopkins University School of Medicine. He has held teaching positions at George Washington Hospital, Yale University, and New York University Medical College. He is now a resident of Oakland where he practices internal medicine. Dr. Sadusk is the author of many papers on the legal aspects of medicine. He is a member of the American Society for Clinical Investigation, the American Federation for Clinical Research, the American Society of Tropical Medicine, the American Public Health Association, the American College of Physicians and the American Heart Association.

Dr. Sadusk will also present a film, "No Margin for Error," dealing with communication failure within the hospital. The film presents some of the information uncovered by Dr. Sadusk recently when he made a survey about professional liability action arising in the hospital. Highlights of the problems the physician faces in the hospital are shown. The film has been very well received by nurses, hospital administrators, and doctors' wives. It is a splendid way of improving human relations within the hospital so that the patient benefits ultimately.

Symposium on Medical Economics

CHAIRMAN: *Robert D. Moreton, M.D., Fort Worth, Texas*

Thursday, November 3, 1960 *9:00 a.m.-12:00 noon*

I. DOCTOR AND HIS OFFICE

THE DOCTOR AS A BUSINESSMAN

John R. Sedgwick, Chicago, Illinois

Consultant on individual and group medical practice for the Sears-Roebuck Foundation.

YARDSTICK FOR ACTION

Woodrow Wirsig, New York, New York

Editor of Printer's Ink, a weekly publication for advertising management, and former Editor of Woman's Home Companion.

THE BUSINESS OF PRACTICING MEDICINE

Horace Cotton, Oradell, New Jersey

A Senior Editor of Medical Economics.

INTERMISSION — VISIT EXHIBITS (30 minutes)

II. DOCTOR AND THE HOSPITAL

STEPS TO PROTECT YOURSELF AGAINST PROFESSIONAL LIABILITY

Joseph F. Sadusk, Jr., M.D., Oakland, California

Member of the American Medical Association Committee on Medical Legal Problems.

NO MARGIN FOR ERROR

Film dealing with communication failure within the hospital.

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topic "Surgical Management of Carotid Insufficiency" he will present some of his extensive experiences in this field, along with follow-up statistics and arteriograms. Dr. Murphey, also an alumnus of Harvard, interned at the Chicago University Clinic. He is Professor of Neurosurgery at the University of Tennessee School of Medicine.

Dr. E. Stanley Crawford, Houston, Texas, will discuss "Surgical Management of Basilar and Carotid Insufficiency Associated with Occlusive Lesions of Aortic Arch and Its Branches." His topic includes clinical recognition and surgical management of the occlusive lesions. Dr. Crawford is a graduate of Harvard Medical School. He interned at Massachusetts General Hospital in Boston and later served as Chief Resident of general and thoracic surgery there. He is now attending surgeon at the Veterans Administration and Methodist Hospitals, both in Houston. Dr. Crawford is also Assistant Professor of Surgery at Baylor University College of Medicine.

Dr. M. M. Keirns will present as his second paper of the morning "Radiological Diagnosis of Intracranial Aneurysms and Vascular Anomalies." He is expected to show how arteriography allows visualization of the underlying pathology and permits formulation of a rational surgical approach for control of intracranial aneurysms.

Dr. Henry G. Schwartz, St. Louis, Missouri, has chosen the topic "Management of Intracranial Aneurysms." The present-day management of aneurysms from the moment of rupture through the definitive surgical attack on the lesion will be presented. Dr. Schwartz is a graduate of Johns Hopkins University School of Medicine. He has taught in the Department of Neurosurgery at Washington University in St. Louis since 1937, and is now Professor in that Department.

Dr. Hubert L. Rosomoff, Pittsburgh, Pennsylvania, is the final speaker of the morning. Under the topic "Hypothermia in Management of Cerebrovascular Lesions" he will discuss how hypothermia protects the brain from damage due to ischemia, anoxia or acute thrombosis or embolism. Dr. Rosomoff graduated from Hahnemann Medical College and Hospital of Philadelphia. He worked for some time at the New York Neurological Institute and is currently on the faculty of the University of Pittsburgh School of Medicine. Dr. Rosomoff has done extensive research on hypothermia and has written numerous articles on that subject.

MEDICAL NEWS

Continued from page 1183

Dr. Benjamin B. Wells, Director of Medical Education Service for the Veterans Administration, has been appointed the VA's Assistant Chief Medical Director for research and education in medicine.

Dr. H. Martin Engle has been named Deputy Chief Medical Director for the VA in Washington.

Dr. Frank Ashburn is a new member of the Southern Society of Clinical Surgeons.

Dr. Morton F. Reiser is President of the American Psychosomatic Society.

Dr. Hewitt I. Varney is President-Elect of the Washington Psychiatric Society. Dr. Leon Yochelson is Secretary; Dr. Morris Kleinerman, Treasurer; and Drs. Paul Chodoff and Henry A. Segal, members of the Council.

Rear Admiral Frank P. Kreuz, Jr., MC, USN, is the new Commanding Officer of the Naval Medical Center.

The Congress of Delegates of the American Academy of General Practice has conferred an honorary membership on Dr. Hugh H. Hussey, Dean of the Georgetown University School of Medicine.

Dr. Henry P. Laughlin will represent the American Psychiatric Association on the Council on Medical Television.

Dr. June C. Shafer has been appointed Chairman of the Committee for Ionic Radiation of the American Academy of Dermatology and Syphilology.

Dr. R. Frank Jones, Medical Director of Freedmen's Hospital, has been appointed by the District Commissioners to the Urban Renewal Council.

Dr. Allan M. McKelvie has been elected First Vice-President of the St. Andrew's Society of Washington.

The Society for Clinical and Experimental Hypnosis will meet at the Willard Hotel, October 6-7. Dr. Lester S. Blumenthal, 5315 Connecticut Avenue, N.W., is the permanent Chairman. For information write the Institute for Research in Hypnosis, 33 East 65th Street, New York 21, New York.

Captain John Atkins, MSC, USA, has been assigned to the Medical Plans and Operations Division, Army Surgeon General's Office, Washington, D. C.

The Board of Trustees of Medical Service of D. C. has re-elected its officers: Dr. James A. Dusbabek,

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Awards of the Southern Medical Association

Do you know of a deserving member of Southern Medical Association whom you would like to nominate for one of the following Awards? If so, there is still time for you to complete the required forms for nomination. However, this should be given your immediate attention. These awards will be presented to the recipients at the Fifty-Fourth Annual Meeting in St. Louis, October 31-November 3, 1960.

DISTINGUISHED SERVICE AWARD

"The Distinguished Service Award of the Association may be awarded annually to any member of the Association in recognition of outstanding contributions to the advancement of medical science. Any member of the Association shall be eligible to receive the award and nominations may be made by any member of the Association."

This award was established in 1955 and has been bestowed upon four outstanding Association members.

RESEARCH MEDAL

"The Research Medal may be awarded from time to time to a member of the Association for meritorious and original research work provided the member has made contributions of sufficient importance to merit this distinction; the Council to provide for a proper committee to evaluate research work and report to the Council."

This award was established in 1912 and has been awarded a total of sixteen times, the last award being made at the Miami Beach meeting in 1957.

SEALE HARRIS MEDAL

"The Seale Harris Medal may be awarded to some member of the Association as recognition for important research accomplishment in the broad field of metabolism, endocrinology, nutrition, or for research which contributes to a better understanding of the chemical changes occurring in disease."

This award, established at the New Orleans meeting in 1958, was awarded for the first time at the Atlanta meeting in 1959.

METHOD OF SELECTION OF RECIPIENT

- Any member of the Association in good standing is eligible for nomination as a recipient of awards.

- Any member in good standing of the Association may nominate a recipient for awards.
- The nominations for the recipient are evaluated by an unpublicized committee which selects three of the nominees annually and submits their names to the Council of the Southern Medical Association.
- The Council elects annually one of the three submitted by the Committee on Special Awards as the recipient.
- The election by the Council is held during the Annual Meeting of the Association and the winner is publicly presented to the Association during the last general session of the membership at a given annual meeting.

MECHANICS

Certain rules and regulations governing mechanics for nomination for awards are provided in the Constitution. Official forms are available from the headquarters office of the Association upon which nominations shall be made. Any member of the Association desiring to place a fellow physician member in nomination may secure official forms from the headquarters office. Forms provide for:

- Biographical information on the nominee, including a recent photograph
- Medical education and training of the nominee
- A professional history, including private practice, specialty training, contributions to medical literature, teaching affiliations, staff connections, etc.
- A detailed description of a specific or general contribution or accomplishment of the nominee to the advancement of medical science or any of its phases upon which the nomination is to be based
- Substantiating evidence of merit, including printed materials, publications, articles, other citations, etc.

The completed forms (and no nomination can be processed without the completion of all proper forms) may be mailed to the Executive Secretary of the Association, 2601 Highland Avenue, Birmingham 5, Alabama, or mailed direct to the First Vice-President. The Committee on Special Awards will submit the names of not more than three nominees for each award to the Council during its first session at a given annual meeting and the election of the recipient by the Council shall be held at its first session.

The elected recipients will be notified of their election and shall receive the award at the last general session of the Association.

For further information, please write:

SOUTHERN MEDICAL ASSOCIATION
2601 Highland Avenue
Birmingham 5, Alabama

Know Your Southern Medical Association

This is the last of a series of seven articles which have appeared in the Journal. They have been presented with the hope that members and friends of the Association may have become better acquainted with the objectives, activities and accomplishments of the Southern Medical Association.

The Headquarters Office

The headquarters office, located in Birmingham, is the nerve center of the Association. It is here that the policies of the Association take on such tangible forms as programs, journals and bulletins. It is here that a multitude of activities carry out the instructions of the governing bodies of the Association.

Day-to-day operations of the office are handled by three operating departments—Membership, Journal, and General Administration.

The Staff

The staff, consisting of twelve employees, include managerial, supervisory, secretarial, clerical and custodial personnel. Each member of the staff is particularly trained for his or her work and the duties and responsibilities of each member are clearly fixed.

The New Building

The activities of the central office are housed in an ultra-modern and especially designed office building owned by the Association. The efficient design and comfortable qualities of the building add greatly to employee efficiency and morale. The total plant value (building, land, and equipment) is in excess of a quarter of a million dollars. The building has a 50% reserve of unfinished office space—adequate for any foreseeable needs.

The Membership Department

The primary activities of this Department include processing new member applications, dues collections, maintaining 95,000 addressograph mailing addresses, handling group insurance, and preparing and mailing membership promotional materials.

The Journal Department

Working closely with the Editor and the Editorial Board, the Journal Department is responsible for the publication of the *Southern Medical Journal* which is owned by the Association. Activities of this Department include proofreading, scheduling, make-up, preparing feature articles and news, sale of reprints and advertising, and mailing 15,000 copies monthly to members and subscribers.

The Administrative Department

Headed by the Executive Secretary, who also exercises supervision over the Journal and Membership Departments, the general administrative affairs are handled by the Administrative Department. Included in the activities of this Department are annual meeting arrangements, publication of programs and the quarterly bulletin, maintenance of minutes and records, services to officers and committees, budgeting, finance and accounting, personnel, general promotion and public relations.

The Purpose

The headquarters office, then, is a workshop dedicated to serving the members of the Association. And, like the Association, it has only one purpose—helping doctors practice better medicine!

V. O. F.

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*New Officers—Elected Atlanta Meeting, 1959.

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MEDICAL NEWS

Continued from page 1188

President; Dr. Henry L. Darner, First Vice-President; and Dr. A. Clagett Gray, Assistant Secretary.

Officers of the Section on General Practice of the Medical Society of the District of Columbia are Dr. Samuel Diener, Chairman; Dr. Lewis A. Klein, Vice-Chairman; and Dr. Charles P. Ryland, Secretary-Treasurer.

Dr. Marc Weiner is Chairman of the Section on Dermatology of the D. C. Medical Society and Dr. James Q. Gant is Secretary.

The Women's Medical Society of the District of Columbia announces the following officers: Dr. Charlotte Donlan, President; Dr. Lois I. Platt, President-Elect; Dr. Alice Chenoweth, Recording Secretary; Dr. Gloria Cochran, Corresponding Secretary; and Dr. Ella F. Andrews, Treasurer.

Dr. Robert H. Parrott has been elected to the Board of Trustees of the National Cystic Fibrosis Research Foundation.

FLORIDA

New members of the Florida State Medical Association include: Dr. Milton G. Abarbanel, West Hollywood; Dr. Theodore L. Batchelder, Jacksonville; Dr. Robert E. Cassidy, Miami Shores; Dr. Mario Fernandez, Miami; Drs. Noel R. Gladson, Edgar W. Gurganious, Jr., and Forrest M. Haswell, all of Jacksonville; Dr. Saul C. Holtzman, St. Petersburg; Dr. Treadwell L. Ireland, Pompano Beach; Dr. Walter G. Jarrell, Jacksonville; Drs. Alfred D. Koenig and Arnold H. Orlick, both of St. Petersburg; Dr. Franklin B. Ott, Pompano Beach; Dr. James A. Parr, St. Petersburg; Dr. Charles E. Pavlik, West Palm Beach; Dr. Robert C. Sheahan, Deerfield Beach; Dr. Ethan O. Todd, Jr., Jacksonville; Dr. Ambrose G. Updegraff, St. Petersburg; Dr. Newton D. Ware, Jacksonville; Dr. Albert H. Wilkinson, Jr., Jacksonville; Dr. Walter A. Guensch, Clearwater; Drs. Harvey Brown, Jr., Harry E. Danielson, Jr., Henry Lubow, and Mark W. Wolcott, all of Coral Gables; Dr. George D. DeLaughter, Jr., Daytona Beach; Dr. William F. Reavis, Eustis; Drs. John B. Britton and B. Joe Wilder, both of Fernandina Beach; Drs. Samuel L. Betagole, Sanford Cobb, Jacob Colsky, Thomas M. Doody, Robert E. Handte, Charles F. Lester, Antonio H. Marquez, Harold E. Register, Harold P. Rizika, and Fred Wasserman, all of Miami; Drs. Jerome G. Block, Eugene A. DeFelice, Eugene Fierer, and Joseph H. Mezer, all of Miami Beach; Drs. Seymour H. Fine, Billy F. Ponder, and Charles R. Sherman, all of North Miami; Dr. Robert S. Darrow, New Port Richey; Dr. Howard S. Hudson, St. Petersburg; Drs. August V. Belott, Byron E. Besse, Jr., William G. Eckert, Roy P. Finney, Jr., William A. Moore III, Elizabeth K. Rockwell, and Sorrell L. Wolfson, all of Tampa; Dr. Argin A. Boggus, Jr., Tavares; Dr. Harold F. Albert, Titusville; Dr. Joseph C. Brettner, Jr., St. Petersburg; Dr. Marcus F. Brown, New Port Richey; Dr. Laurette M. Bryan, Cocoa; Dr. Thomas A. Campbell, St. Petersburg; Dr. Barbara C. Carlton, Wauchula; Dr. McKinley Cheshire, Jr., DeFuniak Springs; Dr. Charles E. Curtiss, Winter Haven; Dr. Daniel L. Donovan, Indialantic; Dr. Gerald L. Ehringer, Daytona Beach; Dr. Freeman Epes, Sarasota; Dr. Michael J. Foley, Melbourne; Drs. Robert L. Gillett and Robert P. Giordano, Sarasota; Dr. W. Roy Hancock, Jacksonville; Dr. Gerald M. Hollingsworth, Ft. Walton Beach; Dr. Minas Joannides, St. Petersburg; Drs. William M. Hutchinson, and William B. Lyle, Jr., Jacksonville; Dr. Frederick Patry, Bradenton; Dr. Jack H. Purcell, St. Petersburg; Dr. James L. Reeder, Fernandina Beach; Dr. Leo E. Reilly, St. Petersburg; Dr. Lamar Roberts, Gainesville; Dr. Hampton L. Schofield, Jr., Vero Beach; Dr. John J. Shinner, St. Petersburg; Drs. Charles R. Smathers, William J. Spann, and Wade N. Stephens, Jacksonville; Dr. Albert Strauss, West Palm Beach; Dr. W. Jape Taylor, Gainesville; Dr. Launey J. Thomas, Jr., Sarasota; Dr. Fred C. Tucker, Clearwater; and Dr. Augustine S. Weekley, Jr., Tampa.

GEORGIA

Dr. Simone Brocato has been installed as President of the Muscogee Medical Society. Other officers include: Dr. Edgar Horn, President-Elect; Dr. Bruce Newsome, Secretary-Treasurer; and Dr. Jack McGee, a member of the Board of Censors. All of the doctors are from Columbus.

Officers of the Southwest Georgia Medical Society are Dr. Homer Woods, Fort Gaines, President; Dr. David Weatherby, Fort Gaines, Vice-President; Dr. J. B. Martin, Edison, Secretary-Treasurer.

Dr. H. Edwin Aderholt, Tifton, has been elected Chief of the Tift County Hospital medical staff.

Dr. John B. Morton, Thomasville, has been made a Diplomate of the American Board of Pediatrics.

Dr. Floyd Sanders is the new President of the DeKalb Medical Society. Other officers include Dr. L. C. Buchanan, President-Elect; Dr. Henry Carter, Vice-President; Dr. Robert Gibbs, Secretary-Treasurer; and Dr. G. L. Mitchell, Corresponding Secretary. The physicians are all of Decatur.

Officers of the St. Joseph's Hospital staff in Savannah are Dr. Lawrence S. Bodziner, President; Dr. Irving Victor, Vice-President; and Dr. J. P. Evans, Secretary.

Serving as officers of county medical societies in Georgia are Dr. Katherine Hendry of Blackshear, President, *Ware;* and Dr. Kathryn S. Lovett of Statesboro, Secretary, *Bullock-Candler-Evans.*

Dr. Alvin D. Josephs, Director of the American Red Cross Regional Blood Center in Atlanta, has been named Medical Officer and Blood Program Director for the organization's Southeastern Area with headquarters in Atlanta.

Dr. Winston E. Burdine, Atlanta, has accepted an appointment as chairman of the advisory board of the American Association of Doctors' Nurses.

Dr. Dorothy Jaeger-Lee is serving as a member of the Nominating Committee of the American Medical Women's Association.

Dr. John T. Godwin, Atlanta, is President of the Georgia Association of Pathologists. He is also an Associate Fellow of the Southeastern Surgical Congress.

Dr. J. Frank Walker is Vice-Speaker of the Medical

APPLICATION FOR HOTEL ACCOMMODATIONS
Southern Medical Association
Saint Louis, Missouri
October 31-November 3, 1960

IMPORTANT: The Convention Reservation Bureau will make hotel assignments upon receipt of this official housing application provided that the application is properly filled out and all necessary information is given. In order that a fair distribution of accommodations can be made to the greatest number of members, all rooms will be assigned on a strictly "first come, first served" basis.

INSTRUCTIONS: *(A) Single rooms are limited in number. Please arrange to occupy double bedded or twin bedrooms if possible. No block reservations will be made.*

(B) Be sure to indicate your arrival time in St. Louis. Reservations will be held only until 6:00 p.m. of the day you indicate when you will arrive in St. Louis. Failure to notify the hotel of any last minute change in your arrival time may result in the cancellation of your reservation.

(C) Write the Convention Reservation Bureau, 911 Locust Street, St. Louis, if you wish to cancel the reservation or make any change in your reservation and NOT the hotel to which you were assigned. This will enable them to reassign rooms that have been cancelled.

(D) If the hotels of your choice are unable to accept your reservation, the Convention Reservation Bureau will make as good a reservation as possible elsewhere provided that all hotel rooms available have not already been taken.

Hotel	For one person	For two persons		2-Room Suite Parlor & 1 Bedroom
		Double Bed	Twin Beds	
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DESOTO	5.50- 8.50	7.00- 10.00	8.50- 12.00	17.50
LENNOX	7.50- 13.00	9.00- 14.00	13.00- 13.50	20.50- 35.00
MAYFAIR	7.50- 13.00	9.00- 14.00	13.00- 14.00	21.00- 35.00
PICK-MARK TWAIN	6.50- 9.00	8.50- 11.50	10.00- 12.50	17.50- 27.00
SHERATON-JEFFERSON	8.50- 11.00	12.00- 14.50	13.85- 18.00	28.00- 49.00
STATLER-HILTON	8.00- 11.50	11.00- 14.50	13.00 -15.00	29.50- 36.50
WARWICK	5.50- 8.00	8.50- 12.00	9.50- 12.00	

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Arrival Date.....	.....hour.....a.m.....p.m.
Departure Date.....	.....hour.....a.m.....p.m.

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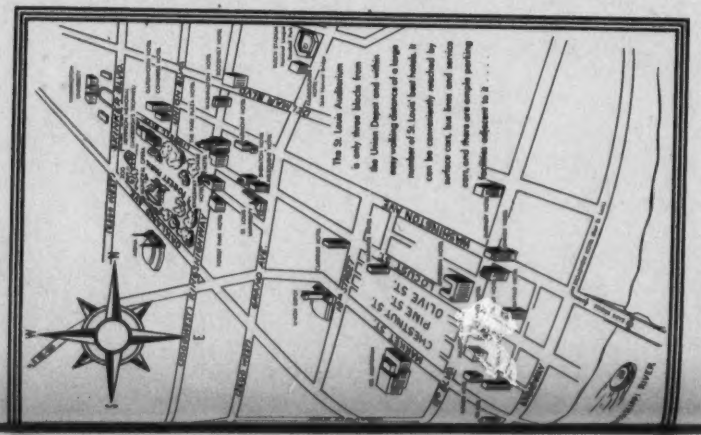
The St. Louis Auditorium is only three blocks from the Union Depot and within easy walking distance of a large number of St. Louis' best hotels. It can be conveniently reached by surface cars, bus lines and service cars, and there are ample parking facilities adjacent to it.
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Association of Georgia House of Delegates and also President of the Georgia Radiological Association.

The following Georgia physicians have recently been certified by the American Board of Obstetrics and Gynecology: Dr. John H. Angell, Savannah, Dr. William S. Boyd, Augusta, Dr. John D. Farris, Waycross, Dr. Richard L. Hanberry, Macon, Dr. Lewis B. Hasty, East Point, Dr. Armand E. Hendee, Atlanta, and Dr. Henry D. Meaders, Marietta.

Dr. Joseph C. Read, Atlanta, is Vice-President of the Southern Society of Clinical Surgeons.

Dr. Hardy A. Kemp has been appointed Manager of the VA Hospital in Atlanta.

KENTUCKY

Dr. J. Duffy Hancock, Louisville, received a special citation for his services to the Social Security Administration and to the medical profession during the meeting of the National Medical Advisory Committee. Dr. Hancock is also Councilor from Kentucky to Southern Medical Association.

Officers of the Kentucky Society of Internal Medicine include Dr. Carl Fortune, Lexington, President; Dr. Robert L. McClendon, Louisville, President-Elect; and Dr. Ralph Denham, Louisville, Secretary-Treasurer.

New members of the Kentucky State Medical Association include Dr. Glenn L. Pfister, Fort Thomas; Dr. R. Burke Suitt, Harlan; and Dr. Paul H. Wilson, Franklin.

Dr. Marc J. Reardon, Covington, is President of the Kentucky Heart Association. Other officers are Dr. Richard R. Crutcher, Lexington, First Vice-President; and Dr. Edmund D. Pellegrino, Lexington, Director.

Dr. William T. Rumage has been elected to a three year term as Chairman of Louisville's Community Chest Council.

Officers of the Kentucky Pediatric Society are Dr. J. J. Glaboff, Louisville, President; Dr. Murvel C. Blair, Frankfort; and Dr. Edwin Paul Scott, Louisville, Secretary-Treasurer.

Dr. B. F. Brown, Owensboro, Director of the Owensboro-Daviess County Health Department, has been named to direct the new State Health Department laboratories in Frankfort.

Dr. A. D. Butterworth, Murray, has accepted the post of Health Officer of Calloway County.

Dr. John H. Githens, who is now with the University of Colorado Medical School, was recently appointed Chairman of the Pediatrics Department in the School of Medicine of the University of Kentucky by the University's Board of Trustees.

The following faculty members of the University of Louisville School of Medicine have been advanced to full Professors of Surgery: Drs. David P. Hall, Alvin B. Ortner, and George B. Sanders.

MISSOURI

Dr. James F. O'Malley is President of the Kansas City Urology Society. Other officers include Dr. C. Laurence Johnson, Vice-President and Dr. Milton B. Ozar, Secretary-Treasurer. The physicians are all of Kansas City.

The Missouri Diabetes Association has elected Dr. Robert L. Jackson, Columbia, President; and Dr. John K. Caldwell, Kansas City, Vice-President. Dr. Thomas W. Burns, Columbia, is Secretary-Treasurer.

Dr. Henry E. Oppenheimer, St. Louis, was recently elected a member of the Council for a three year term by the American Diabetes Association.

The Social Health Society of Greater St. Louis has elected Dr. M. Norman Orgel, St. Louis, as President.

Dr. Vencel W. Hollo, St. Louis, is Chairman of the Medical and Surgical Section of the Association of American Railroads.

Six Washington University School of Medicine faculty members have been promoted to full professorships. Dr. Walter P. Covell is Professor of Anatomy and Professor of Otolaryngology; Dr. John E. Hobbs, Professor of Clinical Obstetrics and Gynecology; Dr. F. Edmund Hunter, Jr., Professor of Pharmacology; Dr. Joseph H. Ogura, Professor of Otolaryngology; Dr. Jack L. Strominger, Professor of Pharmacology; and Dr. Sidney F. Velick, Professor of Biological Chemistry.

Officers of the Kansas City Chapter of the Mis- A. O'Connell, President; Dr. I. C. Fowler, President-Elect; Dr. Robert Butcher, Secretary; and Dr. Richard Owens, Treasurer, all of Kansas City.

New members of medical societies in Missouri include: *Greene County,* Dr. Doris C. Gilpin, Springfield; *Jackson County,* Dr. George K. Boyd, Kansas City, Dr. James W. Vaughn, Kansas City, and Dr. William W. Woodward, Independence; *St. Louis County,* Dr. Albert A. MacPhail, St. Louis; and *St. Louis,* Drs. Robert C. Ahlvin, Dick G. Ellis, Neil I. Gallagher, George E. Magaletta, Marvin H. Marx, Robert M. Weiss, and Kirkley R. Williams, all of St. Louis, and Dr. H. B. Zwirn, Florissant.

Dr. E. Lee Dorsett, University City, is Chief of Staff at St. Louis County Hospital and Dr. Robert Kingsland, Clayton, is Associate Chief of Staff.

NORTH CAROLINA

The University of North Carolina School of Medicine has two news items concerning members of its staff. Dr. Ernest H. Wood, Professor of Radiology, has been elected Vice-President of the American Board of Radiology. Dr. Nathan A. Womack, Head of the Department of Surgery, is President of the National Board of Medical Examiners and President of the United Medical Research Foundation.

New members of the Medical Society of the State of North Carolina include Dr. Bernard L. Richards, Mocksville; Dr. Joachim D. Geratz, Chapel Hill; Dr. Edward B. Mabry, Burlington; Dr. Edgar A. Dillard, Jr., Chapel Hill; Dr. Barbara S. Lipton, Chapel Hill; Dr. Sara J. Dent, Durham; Dr. William C. North, Durham; Dr. William A. Leonard, Greensboro; Dr. Robert L. Phillips, Greensboro; Dr. Powell G. Fox, Jr., Raleigh; Dr. William W. Allen, Pinehurst; Dr.

Continued on page 66

BOOKS RECEIVED

Neurochemistry of Epilepsy. By Donald B. Tower, M.D., Chief of the Section of Clinical Neurochemistry, National Institute of Neurological Diseases and Blindness, Bethesda, and Associate Professor of Neurology, Georgetown University Medical School. American Lecture Series. 290 pages. Springfield, Ill.: Charles C. Thomas, Publisher, 1960. Price $9.00.

Carter's Microbiology and Pathology. By Alice Lorraine Smith, M.D., Professor of Pathology, Graduate Research Institute of Baylor University, Dallas. Seventh edition. 316 illustrations, 696 pages. St. Louis: The C. V. Mosby Company, 1960. Price $7.50.

A Textbook of Gynecology. By Laman A. Gray, M.D., Associate Professor of Obstetrics and Gynecology, University of Louisville Medical School. 458 pages. Springfield, Ill.: Charles C. Thomas, Publisher, 1960. Price $15.50.

Treatment of Urinary Lithiasis. Edited by Arthur J. Butt, M.D., Baptist Hospital, Pensacola, Fla. 512 pages. Springfield, Ill.: Charles C. Thomas, Publisher, 1960. Price $21.00.

Shapes of Sanity. By Ainslie Meares, M.D. 466 pages. Springfield, Ill.: Charles C. Thomas, Publisher, 1959. Price $13.50.

Water Supply for Rural Areas and Small Communities. By Edmund G. Wagner, Chief Engineer and Associate Chief of Field Party, Division of Health and Sanitation, Institute of Inter-American Affairs, Rio de Janeiro, Brazil; and J. N. Lanoix, Sanitary Engineer, WHO, Geneva, Switzerland. 331 pages. WHO Monograph Series No. 42. New York: Columbia University Press, 1959. Price $6.75.

Gestation. Transactions of Fifth Conference, March, 1958, Princeton, N. J. Edited by Claude A. Villee, Ph.D., Harvard Medical School. 247 pages. New York: Josiah Macy, Jr. Foundation, 1959. Price $5.75.

The Practice of Clinical Child Psychology. By Alan O. Ross, Ph.D., Chief Psychologist, Pittsburgh Child Guidance Center. 271 pages. New York: Grune & Stratton, Inc., 1959. Price $5.75.

The Placenta and Fetal Membranes. Edited by Claude A. Villee, Ph.D., Harvard Medical School. 404 pages. Baltimore: Williams & Wilkins Company, 1960. Price $10.00.

A Pharmacologic Approach to the Study of the Mind. Edited by Robert M. Featherstone, Ph.D., Professor and Chairman of the Department of Pharmacology, University of California School of Medicine; and Alexander Simon, M.D., Professor and Chairman of the Department of Psychiatry, University of California School of Medicine. 399 pages. Springfield, Ill.: Charles C. Thomas, Publisher, 1959. Price $10.75.

Diseases of the Chest Including the Heart. Edited by J. Arthur Myers, M.D., Professor of Internal Medicine and Public Health, University of Minnesota. 978 pages. Springfield, Ill.: Charles C. Thomas, Publisher, 1959. Price $34.50.

Pathology of the Heart. Edited by S. E. Gould, M.D., Professor of Pathology, Wayne State University College of Medicine and University of Detroit School of Dentistry. Second edition. 1,114 pages. Springfield, Ill.: Charles C. Thomas, Publisher, 1959. Price $32.50.

Dreams and Personality Dynamics. Edited by Manfred F. De Martino, M.A., School Psychologist, Onondaga County, N. Y. 370 pages. Springfield, Ill.: Charles C. Thomas, Publisher, 1959. Price $10.50.

Cinefluorography. Proceedings of First Annual Symposium, University of Rochester School of Medicine and Dentistry, November 1958. Edited by George H. S. Ramsey, M.D., James S. Watson, Jr., M.D., Theodore A. Tristan, M.D., Sydney Weinberg, and William S. Cornwell, M.A., all from the Department of Radiology, University of Rochester. 262 pages. Springfield, Ill.: Charles C. Thomas, Publisher, 1959.

Immunological and Haematological Surveys. Report of Study Group. WHO No. 181 Technical Report Series. 35 pages. New York: Columbia University Press, 1959. Price $.50.

Rheumatic Fever. Epidemiology and Prevention. Proceedings of a Seminar in Paris in 1956. Edited by R. Cruickshank, M.D., Ch.B., F.R.C.P., D.P.H., and A. A. Glynn, M.B., B.S., M.R.C.P. 191 pages. Springfield, Ill.: Charles C. Thomas, Publisher, 1959. Price $5.50.

The Treatment of Bronchial Neoplasms. By Robert R. Shaw, M.D., and Donald L. Paulson, M.D. 121 pages, No. 3 of The John Alexander Monograph Series. Springfield, Ill.: Charles C. Thomas, Publisher, 1959. Price $8.00.

A Bibliography of Internal Medicine—Selected Diseases. By Arthur L. Bloomfield, M.D., Professor of Medicine, Emeritus, Stanford University School of Medicine. 312 pages. Chicago: The University of Chicago Press, 1960. Price $6.00.

Surgical Gastroenterology. By Warner F. Bowers, M.D., Chief, Department of Surgery, U. S. Army Hospital, Honolulu. 492 pages. Springfield, Ill.: Charles C. Thomas, Publisher, 1960. Price $18.50.

The Human Apocrine Sweat Gland in Health and Disease. By Harry J. Hurley, M.D., Professor of Dermatology, Hahnemann Medical College; and Walter B. Shelley, M.D., Professor of Dermatology, University of Pennsylvania School of Medicine, both of Philadelphia. 134 pages. Springfield, Ill.: Charles C. Thomas, Publisher, 1960. Price $6.50.

Medical Care of the Adolescent. By J. Roswell Gallagher, M.D., The Children's Hospital Medical Center, Boston. 360 pages. New York: Appleton-Century-Crofts, Inc., 1960.

Surgery of Repair as Applied to Hand Injuries. By B. K. Rank, C.M.G.; and A. R. Wakefield, M.S. (Melbourne), F.R.C.S. (Eng.) F.R.A.C.S. Second edition. 277 pages. Baltimore: The Williams & Wilkins Company, 1960. Price $9.00.

Myocardosis. By Ferdinand Wuhrmann, M.D., Chief, Medical Service, Cantonal Hospital of Winterthur, Switzerland. 216 pages. Springfield, Ill.: Charles C. Thomas, Publisher, 1960. Price $10.50.

Fellowship of Surgeons. By Loyal Davis, M.D. 507 pages. Springfield, Ill.: Charles C. Thomas, Publisher, 1959. Price $10.50.

Surgical Anatomy of the Bronchovascular Segments. By William E. Bloomer, M.D., Assistant Professor of Surgery, Averill A. Liebow, M.D., John Slade Ely, Professor of Pathology, and Milton R. Hales, M.D., Assistant Professor of Pathology, all of Yale University. 270 pages. Springfield, Ill.: Charles C. Thomas, Publisher, 1960. Price $16.50.

Basic Facts of Body Water and Ions. By Stewart M. Brooks, M.S., Science Instructor, Lasell Junior College, Auburndale, Mass. 152 pages. New York: Springer Publishing Company Inc., 1960.

Medical, Surgical, and Gynecological Complications of Pregnancy. Edited by Alan F. Guttmacher, M.D., Obstetrician and Gynecologist-in-Chief; and Joseph J. Rovinsky, M.D., Assistant Attending Obstetrician and Gynecologist, both of the Mount Sinai Hospital, N. Y. 604 pages. Baltimore: The Williams & Wilkins Company, 1960. Price $16.50.

Essentials of Fluid Balance. By D. A. K. Black, M.D., F.R.C.P., Professor of Medicine, University of Manchester. Second edition. 132 pages. Springfield, Ill.: Charles C. Thomas, Publisher, 1960. Price $4.50.

Thymectomy for Myasthenia Gravis. By Henry R. Viets, M.D., Lecturer on Neurology, Emeritus, Harvard Medical School, and Robert S. Schwab, M.D., Assistant Clinical Professor of Neurology, Harvard Medical School. 130 pages. Springfield, Ill.: Charles C. Thomas, Publisher, 1960. Price $7.00.

Physiology of Prematurity. Transactions of the Fourth Conference, March, 1959, Princeton, N. J. Edited by Jonathan T. Lanman, M.D., Department of Pediatrics, New York University-Bellevue Medical Center, New York. 176 pages. New York: Josiah Macy, Jr., Foundation, 1960. Price $4.50.

Recent Advances in Public Health. By J. L. Burn, M.D., D.Hy., D.P.H., Lecturer in Public Health, University of Manchester. Second edition. 370 pages. Boston: Little, Brown & Company, 1960. Price $10.00.

Basic Office Dermatology. By Stuart Maddin, M.D., Vancouver General Hospital; Julius L. Danto, M.D., Vancouver General Hospital; and William D. Stewart, M.D., Department of Medicine, University of British Columbia. 296 pages. Springfield, Ill.: Charles C. Thomas, Publisher, 1960. Price $11.75.

Advances in Gynecologic Surgery. Clinical Obstetrics and Gynecology. Vol. 2, No. 4. New York: Paul B. Hoeber, Inc., 1960. Price $18 per year.

Encyclopedia of Medical Syndromes. Robert H. Durham, M.D., Physician-in-Charge, Division of General Medicine, Henry Ford Hospital, Detroit. 613 pages. New York: Paul B. Hoeber, Inc., 1960. Price $13.50.

Diabetes. Edited by Robert H. Williams, M.D., Professor of Medicine, University of Washington. 774 pages. New York: Paul B. Hoeber, Inc., 1960. Price $20.00.

X-ray Technology. By Charles A. Jacobi, B.Sc., R.T. and M.T., Chairman, Medical X-ray Technology, Oregon Technical Institute; and Donald E. Hagen, R. T., Portland, Oregon. Second edition. 440 pages, 320 illustrations. St. Louis: The C. V. Mosby Company, 1960. Price $10.00.

Research Conference on Therapeutic Community. Held at Manhattan State Hospital. Edited by Herman C. B. Denber, M.D., Instructor in Psychiatry, College of Physicians and Surgeons, Columbia University. 245 pages. Springfield, Ill.: Charles C. Thomas, Publisher, 1960. Price $11.00.

Interpersonal Relationships in the Hospital. By Warner F. Bowers, M.D. 125 pages. Springfield, Ill.: Charles C. Thomas, Publisher, 1960. Price $5.00.

New Methods of Studying Gaseous Exchange and Pulmonary Function. By Alfred Fleisch, M.D., Professor of Lausanne University, Switzerland, translated by Charles Corsi. Third edition. 116 pages. Springfield, Ill.: Charles C. Thomas, Publisher, 1960. Price $5.75.

Enzymes in Health and Disease. Edited by David M. Greenberg, Ph.D., Professor of Biochemistry and Chairman of the Department, University of California School of Medicine; and Harold A. Harper, Ph.D., Professor of Biochemistry, University of California School of Medicine. 459 pages. Springfield, Ill.: Charles C. Thomas, Publisher, 1960. Price $14.50.

Textbook of Otolaryngology. By David D. DeWeese, M.D., Clinical Professor of Otolaryngology, University of Oregon Medical School; and William H. Saunders, M.D., Associate Professor of Otolaryngology, the Ohio State University College of Medicine. 455 pages, with 354 illustrations. St. Louis: The C. V. Mosby Company, 1960. Price $8.75.

Atlas of Anatomy and Surgical Approaches in Orthopaedic Surgery—Upper Extremity. By Rudolfo Cosentino, M.D., Assistant Professor in Orthopaedic Surgery, University of La Plata, Argentina, and Research Associate, Department of Orthopaedic Surgery, State University of Iowa. 192 pages, illustrated. Springfield, Ill.: Charles C. Thomas, Publisher, 1960. Price $10.50.

Chemical Micromethods in Clinical Medicine. By R. H. Wilkinson, M.D., Assistant Chemical Pathologist, The Hospital for Sick Children, London. American Lecture Series. 110 pages. Springfield, Ill.: Charles C. Thomas, Publisher, 1960. Price $5.00.

A Stereotaxic Atlas of the Dog's Brain. By Robert K. S. Lim, Ph.D., D.Sc., and Robert L. Moffitt, B.S., both of Miles Laboratories, Inc., Elkhart, Indiana; and Chan-Nao Liu, Ph.D., Department of Anatomy, University of Pennsylvania. 93 pages. Springfield, Ill.: Charles C. Thomas, Publisher, 1960.

Massage, Manipulation and Traction. Edited by Sidney Licht, M.D. Fifth volume of Physical Medicine Library. 258 pages. New Haven, Conn.: Elizabeth Licht, Publisher, 1960. Price $10.00.

Photography in Medicine. By Arthur Smialowski, Director, Department of Photography, St. Michael's Hospital, Toronto; and Donald J. Currie, M.D., Clinical Teacher, Department of Surgery, University of Toronto. 316 pages. Springfield, Ill.: Charles C. Thomas, Publisher, 1960. Price $14.50.

Studies on Vertebrate Neurogenesis. By Professor Santiago Ramon Y. Cajal, Faculty of Medicine, University of Madrid. Translated by Lloyd Guth, M.D., National Institutes of Health, Bethesda. 423 pages. Springfield, Ill.: Charles C. Thomas, Publisher, 1960. Price $13.50.

Radiologic Records. By Sister Christina Spirko, C.S.J., M.A., Department of Roentgenology, St. Mary's Hospital, Amsterdam, N. Y. 293 pages. Springfield, Ill.: Charles C. Thomas, Publisher, 1960. Price $8.50.

Oxygen Supply to the Human Fetus. Symposium by Council for International Organizations of Medical Sciences and Josiah Macy, Jr., Foundation. Edited by James Walker and Alex C. Turnbull. 306 pages. Springfield, Ill.: Charles C. Thomas, Publisher, 1959. Price $10.50.

Communicable and Infectious Diseases. By Franklin H. Top, M.D., Professor and Head, Department of Hygiene and Preventive Medicine, State University of Iowa. Fourth edition. 768 pages. St. Louis: The C. V. Mosby Company, 1960. Price $20.00.

Polysaccharides in Biology. Transactions of the Fourth Conference, May, 1958. Edited by Georg F. Springer, M.D., University of Pennsylvania School of Medicine. 306 pages. New York: Josiah Macy, Jr., Foundation, 1959. Price $5.95.

Cancer of the Cervix. Diagnosis of Early Forms. Ciba Foundation Study Group No. 3. Edited by G. E. W. Wolstenholme and Maeve O'Connor. 110 pages. Boston: Little, Brown & Company, 1959. Price $2.50.

Principles of Orthopaedic Surgery. By Paul C. Colonna, M.D., Professor of Orthopaedic Surgery, Emeritus, University of Pennsylvania Medical School. 783 pages. Boston: Little, Brown & Company, 1960. Price $22.00.

Glaucoma. Transaction of Fourth Conference, March, 1959. Edited by Frank W. Newell, M.D., Department of Surgery (Ophthalmology). 244 pages. New York: Josiah Macy, Jr., Foundation, 1960. Price $8.00.

Significant Trends in Medical Research. Ciba Foundation, Tenth Anniversary Symposium. Edited by G. E. W. Wolstenholme, Cecilia M. O'Connor and Maeve O'Connor. 335 pages. Boston: Little, Brown & Company, 1959. Price $9.50.

Biochemistry of Human Genetics. Ciba Foundation Symposium. Edited by G. E. W. Wolstenholme and Cecilia M. O'Connor. 328 pages. Boston: Little, Brown & Company, 1959. Price $9.50.

Baby Name Finder. By J. E. Schmidt, M.D. 390 pages. Springfield, Ill.: Charles C. Thomas, Publisher, 1960. Price $10.50.

INSTRUCTIONS TO CONTRIBUTORS

Exclusive Publication: Articles offered for publication must be contributed solely to the Southern Medical Journal.

Manuscripts: Manuscripts should be original copy, typewritten, double-spaced, with wide margins. Because of lack of space, it is necessary to limit the number of bibliographic references to twenty. Footnotes and references should conform to the following style:

1. Doe, J. E.: What You Should Know about It, South. M. J. 50:711, 1950.

Illustrations: Black and white glossy prints, preferably 5 by 7 inches, and drawings in India ink on white paper are required. The author's name, number and indication of top, if doubtful, should be attached. Necessary plates not exceeding six, or one and a half pages, will be furnished by the Journal.

Reprints: Reprints are available at publisher's cost. An order form will accompany author's galley proof and should be returned with the galley to the Editor.

Book Reviews: Books and monographs submitted for review should be mailed to the Editor. Acknowledgment will appear in the Journal. Selection rights reserved.

Editorial Address: All manuscripts, corrected galley proof, reprint orders, books for review and related correspondence should be addressed to the Editor, R. H. Kampmeier, M.D., Vanderbilt University School of Medicine, Nashville 5, Tennessee.

Business Address: All correspondence related to membership, subscriptions, advertising, and other business should be addressed to the Southern Medical Association, 2601 Highland Avenue, Birmingham 5, Alabama.

BOOK REVIEWS

The Process of Aging in the Nervous System

Edited by James E. Birren, Ph.D., Henry A. Imus, Ph.D., and William F. Windle, Ph.D. 217 pages. Springfield, Ill.: Charles C. Thomas, Publisher, 1959. Price $7.00.

This monograph is the fifth in a series of "Symposia in Neuroanatomical Sciences." There is a distinguished list of contributors. The book includes their papers as well as the discussions of the conference on "The Process of Aging in the Nervous System" which was held in early 1957.

This very important subject of aging is considered from many aspects, including the regeneration, histochemistry, physiology, biochemistry and, finally, the pathologic changes of the nervous system. Some of the more recent observations based on electron microscopy are included. Again, one of the most commendable features of these monographs is the reproduction of round-table discussions.

This work is highly recommended to all workers who have an interest in the neuroanatomic sciences as well as to neurologists, neurosurgeons and neuropathologists. It should be in every medical library.

Anastomoses Between the Leptomeningeal Arteries of the Brain

By Henri M. Vander Eecken, M.D., Professor of Medical Psychology and Agrege in Neurology, University of Ghent, Belgium. 154 pages. Springfield, Ill.: Charles C. Thomas, Publisher, 1959. Price $7.50.

This monograph is a noteworthy contribution to our knowledge of the vasculature of the brain. Its publication is timely in view of the current resurgence of interest in cerebrovascular disease. The problem of leptomeningeal anastomoses has received relatively little attention during the past half century, and further study of this subject is relevant if one is to explain the variable pathologic and clinical pictures associated with occlusion of cerebral vessels.

The first chapter of this volume deals in detail with the cerebral blood supply and its intercommunications. In the second chapter there is detailed an anatomic study of the extent and distribution of the leptomeningeal anastomoses. This is supplemented with the results of embryologic and comparative anatomic studies. The physiologist, clinician and neuropathologist will be interested in the final chapters in which the functional significance of this problem is assessed and in which clinical neuropathologic studies are reviewed. This work is well illustrated and includes an excellent bibliography. This volume will be of great interest to those interested in the basic neuroanatomic sciences and in their clinical applications.

Neurology

By Roy R. Grinker, M.D., Director of Institute for Psychosomatic and Psychiatric Research and Training; Paul B. Bucy, M.D., Professor of Surgery, Northwestern University School of Medicine; and Adolph L. Sahs, M.D., Professor and Head of Department of Neurology, State University of Iowa. Fifth edition. 1,296 pages. Springfield, Ill.: Charles C. Thomas, Publisher, 1960. Price $24.50.

This well-known textbook of neurology, which has been available for the past 25 years, has now been completely revised and expanded with the assistance of the eminent co-authors of this edition. The new section on neurochemistry and metabolic diseases is a very welcome innovation. During the preparation of this section, which constitutes a very readable account of some fundamental aspects of neurochemistry, the authors have been fortunate in the advice and guidance of Dr. Donald Tower. The chapter on trauma to the central nervous system is complete and up-to-date with descriptions of current therapeutic concepts regarding subdural hematoma in infants, postmeningitic subdural effusions and infantile hemiplegia.

This textbook is highly recommended to medical students, residents and physicians who are interested in clinical neurology and the related fields. The present edition represents a considerable improvement and expansion in what has always been regarded as an excellent textbook.

Diseases of the Nervous System in Infancy, Childhood and Adolescence

By Frank R. Ford, M.D., Johns Hopkins Hospital, Baltimore. Fourth edition. 1,326 pages. Springfield, Ill.: Charles C. Thomas, Publisher, 1960.

This monumental work has been greatly expanded. As the publishers indicate, this revision is most complete. To the section on infectious diseases such viral disorders as coxsackie infections, encephalomyocarditis and cytomegalic inclusion disease have been added. The chapter on toxic and metabolic diseases has been enlarged to include the neurologic manifestations associated with a variety of poisons and venoms as well as some of the disorders which are of current interest because of their neurochemical implications. The latter include galactosuria, familial methemoglobinemia, familial amyloidosis and hypophosphatasia. The encyclopedic scope of this volume is indicated by the detailed index which comprises more than 200 pages.

The author and the publishers deserve high praise for this new and enlarged edition. Pediatricians, neurologists and others interested in the neurologic disorders of childhood will find this a very useful reference work. Many who possess earlier editions of this work will wish to replace them with this more complete and up-to-date fourth edition.

Rudolph Matas History of the Louisiana State Medical Society

Edited by Mary Louise Marshall. 2 Volumes, each 239 pages. New Orleans, La.: Rudolph Matas History of Medicine Trust Fund, 1957.

These two volumes represent the original aim for

which Dr. Rudolph Matas began to collect historical material 34 years ago, when he was entrusted with the task of preparing a history of the Louisiana State Medical Society. The material finally collected, however, went far beyond the initial scope and now embraces the whole of the history of medicine of Louisiana from its earliest beginnings. It is deposited in the Rudolph Matas Medical Library of Tulane University, where it is available to researchers.

The first volume of this history, edited by Miss Mary Louise Marshall, librarian of the Rudolph Matas Medical Library, follows the rules laid down by Dr. Matas; he had felt that a group can best be known through its exponents. "For this reason a biographic study of the presidents of the Louisiana State Medical Society is of interest and importance in connection with the chronological record of its proceedings and transactions, as a guide to the policies and objectives that prompt the electors in their selection and also in estimating the achievements accomplished by the elect during their terms of office," so had written Dr. Matas. The biographical sketches with their wealth of information are accompanied by excellent photographs and represent all of the presidents of the Society since its inception in 1878.

But from the pages of this book also emerges the unique situation of medicine in Louisiana. The introductory chapter by Professor John Duffy shows the importance of this work also to the cultural historian of this country. One reason for the late coming into being of a state medical society in Louisiana was the rift between the French and American physicians. Rival societies were formed and feuded with each other. But from the standpoint of medicine this dual cultural system had some advantages. In the colonial period the surgeons and physicians were nearly all trained in Europe, at a time when France was outstanding in the field of surgery. French schools emphasized training in the humanities, which gave the Creole physicians a broader general outlook and prepared them better for intellectual leadership in their community. On the other hand, the pragmatic approach of the American-trained medical men left them freer from the restraints of fixed medical theories and more apt to observe and experiment. In Louisiana these two schools of medicine were brought into close conjunction. Contact between the better French and American physicians gradually engendered mutual respect and each respectively leavened the medical practices of the other, so that by 1860 New Orleans was rapidly taking Southern leadership in medical education, journalism and other aspects of medicine. The proceedings of the Society are not without humor, as when Miss Marshall relates the coming into being of the Woman's Auxiliary. The physicians' wives were enthusiastic to put their energies to work in the field of public relations and education on behalf of the Medical Society, but there was strong opposition on the part of some of the physicians. After the creation of the Woman's Auxiliary had been sabotaged a number of times, another adverse report was made in 1929. The President of the Society, Dr. Herold, who favored the new organization, urbanely moved that the committee that had presented the adverse report be discharged with thanks, and that the report be filed. And then, apparently the same smooth and urbane manner went on to move that the Society proceed to organize an Auxiliary the following day. It must be assumed that the delegates did not quite fathom what was happening, because the motion was carried without any opposition, to the great surprise of everybody.

Volume II of this history is edited by Mrs. Hathaway Gibbens Aleman, the secretary of Dr. Matas until his end, who assisted him for many years in collecting the encyclopedic material which forms the basis for this work.

It gives the histories of the medical societies of Louisiana which formed independently in the individual parishes and congressional districts, both before and since the creation of the State Medical Society in 1878. Often these local societies did not last very long; many parishes never succeeded in keeping one alive over a couple of years, so that, when, in 1881 the parish of Morehouse organized a society, the editors of the *New Orleans Medical and Surgical Journal* made the following comment:

"We feel a little proud and encouraged, as one might in announcing that a man child is born into the world. There are not many such children in Louisiana, for our physicians have not been happy in this sort of conception; the birth-rate has been low and the death-rate high. We do not suspect any unlawful tricks to prevent conception; the cause must be either infertility from organic defect, or long delayed puberty. Let us trust that it is the latter, and hope for better luck in the future."

The hopes of the editor were realized: 60 years later there were 48 parishes with organized societies, and seven active district medical societies in Louisiana. After World War II there were fewer societies in parishes where the number of physicians was small. Instead, those interested in organized medicine joined the larger medical societies located in cities, or district societies, and only three parishes, with a combined total of six registered physicians had no representation whatsoever.

Sometimes one regrets that the editors have placed a little too much restraint upon themselves, as when Mrs. Aleman tells of the consistent blackballing of women physicians, from 1905 to 1913. One would like to hear a few details about "the bitter struggle" that preceded their being admitted to full membership, as well as to know exactly what "the mythical 'Female Section'" was. Mrs. Aleman still has access to oral tradition which gives her unique opportunities to fill many gaps which later on will remain permanent gaps.

These first two volumes mined from the material collected by Dr. Matas are a splendid beginning, which now is being followed by Professor John Duffy's "The Rudolph Matas History of Louisiana Medicine." Together they constitute a solid monument to Dr. Matas and his dedicated helpers and coworkers.

Synopsis of Treatment of Anorectal Diseases

By Stuart T. Ross, M.D. 235 pages. St. Louis: C. V. Mosby Company, 1959. Price $6.50.

This is another of the well-known C. V. Mosby "Synopsis" series, this time concerning anorectal diseases. The book is a pocket-sized 235 page handbook

which deals with the fundamental facts all physicians and surgeons must possess in order to handle proctologic ailments effectively.

In his foreword, Dr. Harry E. Bacon, an internationally known proctologist, summarizes, "For three decades not a single treatise in manual form has appeared on this subject, and this particular synopsis is a most welcome addition to the library of all interested in this field of scientific endeavour."

As in all the "Synopsis" books an effort is made toward concise presentation with a minimum of superfluity and academic meanderings. This might be criticized by those with some knowledge of the subject, but it is ideal for those who need a quick reference work.

Mitral Valvulotomy

By Harold J. Stewart, M.D., Professor of Clinical Medicine, Cornell University Medical College; and Frank Glenn, M.D., Lewis Atterbury Stimson, Professor of Surgery (Chairman), Cornell University Medical College. 231 pages. Springfield, Ill.: Charles C. Thomas, Publisher, 1959. Price $10.50.

This is a 244 page monograph on the experiences of the Cornell Medical Center Group with the closed technic of mitral valvulotomy from 1951 through April 1956. A thorough analysis of the criteria for surgery, the operative mortality, and the clinical results are presented for 300 patients. The authors admit that such a series should not be considered statistically consequential in itself but add their experience to that rapidly accumulating in the literature. Detailed studies of the immediate and delayed postoperative deaths are presented.

Emphasis is given to the importance of gallbladder disease in these patients. The authors feel that mitral stenosis predisposes to biliary disease. Their policy is to correct biliary disease first, unless valvulotomy is very urgent, and then follow with the chest procedure in three weeks.

Since this is a statistical survey, the clinical aspects of the disease and the details of management are not presented as thoroughly as one would expect in a textbook.

Rheumatic Fever. Epidemiology and Prevention. Proceedings of a Seminar in Paris in 1956

Edited by R. Cruickshank, M.D., Ch.B., F.R.C.P., D.P.H., and A. A. Glynn, M.B., B.S., M.R.C.P. 191 pages. Springfield, Ill.: Charles C. Thomas, Publisher, 1959. Price $5.50.

This book contains the papers presented at a seminar on the epidemiology and prevention of rheumatic fever held at the International Children's Centre in Paris in the early Autumn of 1956. Although nothing new is contained in the report, it is an excellent review and is thoroughly up-to-date, since nothing new has happened since then to add to our knowledge of rheumatic fever.

The majority of the participants were French, but a scattering of representatives from most of the Western Nations gave the seminar a truly international flavor. It is of interest how similar the ideas presented are to those which are prevalent in the U. S. and incidentally it is also of note how many words are misspelled.

A plea was made by several participants for standardization of tests for measuring streptococcal antibodies and for correlation of clinical epidemiologic and serologic studies. Resistance of hemolytic streptococci to penicillin was not reported but 75% of strains of streptococci isolated in Czechoslovakia were sulfonamide-resistant.

There was general agreement that the hemolytic streptococcus is the agent factor in rheumatic fever but, enough differences in the experience of various reporters on the epidemiology of rheumatic fever were expressed to indicate that the environmental factors which are important in one country may not be the same as those in another. For example, in Bologna the disease is particularly widespread in rural areas and the high incidence of rheumatic fever in the training centers in the U. S. Armed Forces during World War II had no parallel in Great Britain. It is refreshing to learn in this day when one can hardly read a clinical journal that does not extol the virtues of penicillin, either in its advertisements or articles, that at least two investigators do not agree entirely that primary attacks of rheumatic fever can be prevented by penicillin therapy.

Pediatricians, medical students and others interested in rheumatic fever will find this little book very useful.

Basic Surgery

Edited by Leslie Oliver, M.B., B.S. (Lond.), F.R.C.S. (Eng.), F.A.C.S., Neurosurgeon, West London Hospital and Medical School. 1,327 pages, 680 illustrations. Springfield, Ill.: Charles C. Thomas, Publisher, 1959. Price $26.50.

This is a very large volume by British authors intended, as the title indicates, to present the most fundamental aspects of surgical diseases for the undergraduate student. There is no bibliography and no real effort to present the practical details of surgical technic.

All specialties in surgery are covered except gynecology and otorhinolaryngology. The photographs are excellent and many simple, clear schematic diagrams are provided.

There is, of course, much basic difference in the British and the American approach toward certain subjects, i.e., fluid and electrolyte balance, the treatment of inguinal hernia, and the surgical therapy of pulmonary tuberculosis. These differences would give the American student a distorted view of these subjects from that he would be likely to encounter in America. The book is good for its ascribed purpose but would not be especially valuable at the postgraduate level.

Therapeutic Electricity and Ultraviolet Radiation

Edited by Sidney Licht, M.D., Honorary Member, British Association of Physical Medicine, Danish Society of Physical Medicine and the French National Society of Physical Medicine. 343 pages, illustrated. New Haven, Conn.: Elizabeth Licht, Publisher, 1959. Price $10.00.

Therapeutic Electricity and Ultraviolet Radiation

is the fourth volume in the Physical Medicine Library. Each of the nine chapters has been written by a specialist in his respective field with five representatives from the United States, two from England and one from Russia. The purpose of the editor was to bring together under one cover all the information, historical and current, on therapeutic electricity and ultraviolet radiation. The actual value of electrotherapy is of questionable importance but the number of therapeutic devices manufactured and sold remains high and there is a continued demand for electrotherapy; therefore, there is a definite need for current information on the subject.

As indicated by the title, the book is composed of two sections. Under Therapeutic Electricity, the following topics are presented: History of Electrotherapy, Instrumentation for Electrotherapy, Clincial Electrical Stimulation, Iontophoresis and Electrosleep Therapy.

The Ultraviolet Radiation section covers History of Ultraviolet Therapy, Instrumentation for Ultraviolet Therapy, Physiologic Effects of Ultraviolet Radiation, and Clinical Uses of Ultraviolet Radiation.

Dr. Licht has prepared the history of electrotherapy and of ultraviolet therapy and in both cases, the histories are most thorough. The bibliographies show the exhaustive extent of the studies with 923 references on electrotherapy and 85 on ultraviolet therapy. Each contributor has also presented his complete topic in great detail. Each has his own illustrations and his own list of references. Indices of authors cited and of subjects have been included. Diathermy, ultrasonic therapy and other forms of therapeutic heat have been presented in Volume II of the series and are not considered in this book.

Since this is the most complete and up-to-date presentation of the entire subject of both electrotherapy and ultraviolet therapy, the book will be helpful to those teaching or studying this particular subject matter and to those who will wish to use it as a reference book.

Acute Cardiac Pulmonary Edema

By Sigmund Wassermann, M.D., formerly with the First Medical Clinic, Vienna, Austria. 123 pages. Springfield, Ill.: Charles C. Thomas, Publisher, 1959. Price $4.25.

This is a small monograph on a subject in which the author has been interested for a lifetime. Dr. Wassermann presents a scholarly essay which thoroughly analyzes all aspects of the problem. He presents and refutes the older concept of the mechanical basis for pulmonary edema and presents the newer reflex basis to which he subscribes. The bibliography is comprehensive. The work is limited in scope but beautifully done.

Progressive Exercise Therapy in Rehabilitation and Physical Education

By John H. C. Colson, F.C.S.P., M.S.R.G., M.A.O.T., Wakefield, England. 184 pages. Baltimore: Williams & Wilkins Company, 1958. Price $4.50.

The author deals with the subject progressive exercise therapy on the basis of his 20 years of experience in hospital, rehabilitation and private clinic situations. The book presents a collection of exercises which are graded progressively as to strength, endurance and/or mobility and is divided into three major parts. Section I relates to progression in exercises for each segment of the body; section II pertains to exercise programs for such conditions as gastrectomy, operations for inguinal hernia, and appendectomy, menisectomy and intervertebral disk lesions of the lumbar spine; and section III relates to general group exercise programs adapted to music and "circuit training" which is a new form of physical education stressing progressive development of endurance and strength. An appendix, bibliography and index are given.

In general, all chapters are written in the same format. There is an introductory paragraph relating to the subject matter presented, stick figures are used to illustrate the exercises and progressions are in outline form. To the uninitiated, the English terminology is difficult to follow, but the diagrams used are self-explanatory.

The book is of interest to physical education majors, physical therapists and remedial gymnasts. It is probably more meaningful to those working in the group situation and the longer, convalescent treatment phase. As in any book which presents the author's skills or technics, there are many areas open for discussion. It is obvious that the author has had many years of experience in the treatment area of exercise.

The Clinical Evaluation of New Drugs

Edited by S. O. Waife, M.D., and Alvin P. Shapiro, M.D. 216 pages. New York: Paul B. Hoeber, Inc., 1959. Price $7.50.

This monograph on the clinical evaluation of new drugs is one of the most valuable contributions to this subject in recent years. The monograph is divided into two sections: (1) Principles of Drug Evaluation, and (2) Clinical Trials and Practice. Considerable attention is given to good experimental design which is so essential if answers obtained from the research are to be useful and meaningful. This lack of adequate experimental design has been the weakness of many studies on the clinical evaluation of drugs. If an investigator carefully takes into consideration the factors so concisely but yet so clearly defined in this monograph there should be considerable improvement seen in the evaluation of new chemotherapeutic agents. A careful perusal of this monograph before an investigator undertakes a new study may save him many weeks or months of study. This book is highly recommended for the clinical investigator.

The Surgical Treatment of Facial Injuries

By Varaztad Hovhannes Kazanjian, M.D., Professor Emeritus of Plastic Surgery, Harvard University; and John Marquis Converse, M.D., Professor of Plastic Surgery, New York University College of Medicine. Second edition. 1,096 pages. Baltimore: Williams & Wilkins Company, 1959. Price $22.00.

This is a beautifully written encyclopedic work covering every conceivable aspect of the management of facial trauma. The senior author is singularly well qualified to write in this field with an experience of unrivalled breadth covering experiences in both World Wars. Chapters are included on wound healing, all

types of free and pedicle grafts, the roentgenology of the facial bones, the early emergency treatment of facial injuries, the care of facial trauma in children, and maxillofacial prosthetics as well as the detailed step-by-step consideration of reconstruction of tissue deformity in each area of the face.

Clinical management is masterfully illustrated by hundreds of actual photographs as well as schematic diagrams of excellent quality. The book can be recommended as excellent for the plastic surgeon and very worthwhile for the surgeon who must deal with trauma.

A Manual of Electrotherapy

By Arthur L. Watkins, M.D., Assistant Clinical Professor of Medicine, Harvard Medical School. 251 pages, 167 illustrations. Philadelphia: Lea & Febiger, 1958. Price $5.00.

In writing *A Manual of Electrotherapy* the author states that he has drawn freely from the textbook *A Manual of Physical Therapy and Electrotherapy and Light Therapy* by the late Dr. Richard Kovács. Dr. Watkins has been a specialist in the field of Physical Medicine for many years and is currently Chief of this service at Massachusetts General Hospital in Boston.

The book is divided into five parts. Part I contains two short chapters dealing with the scope and history of physical therapy. The author has retained this presentation as written by Dr. Kovács. Part II deals with the physics and physiologic effects of heat and infrared radiation. Equipment for this type of heat and the technics of its application in the clinical situation are described. Part III discusses the thermal and physiologic effects of high and low frequency currents. The types of equipment used for treatment purposes and the technics or application are presented. Part IV relates to electrophysics which includes generation, conversion and distribution of electricity. Part V consists of one short chapter on ultrasound and gives the history, physics, physiology and technic of application. This chapter has been added by the author.

Doctors, medical students and physical therapists interested in electrotherapy and electrodiagnosis will find this book a concise, helpful manual. The style of writing is clear and to the point; chapter content follows the same format throughout with bold captions for paragraph headings and there are numerous, meaningful illustrations.

Neurology of Infancy

By Anatole Dekaban, M.D., Assistant Professor of Neurology, George Washington University Medical School. 382 pages. Baltimore: Williams & Wilkins Company, 1959. Price $12.00.

This volume reflects the current intense interest in neurologic problems of the childhood in general and infancy in particular. It probably will find greater use as a handbook or reference work than as a textbook.

Consideration, of varying length, is given to virtually every condition of infancy which may affect the central nervous system exclusively, or as part of a broader clinical syndrome of which there is a surprisingly large number. The major topics include, among others, birth injuries, vascular disorders, infectious diseases, congenital anomalies, neoplastic and degenerative disorders and metabolic, and endocrine disorders affecting the central nervous system.

Chapters devoted to developmental anatomy and physiology, birth injuries and vascular disorders of the central nervous system are well done and include much information not readily obtainable elsewhere in a single volume. Others, particularly on systemic disease are better handled in more limited monographs or pediatric texts and the problems of congenital anomalies and tumors are more extensively treated in neurosurgical texts.

The illustrations are limited in number but of good quality, particularly photographs of gross anatomic and pathologic specimens.

The index is quite brief and some subjects are difficult to locate. This could be improved upon.

The references, virtually all of which are in English, at the end of the chapters, are in alphabetical order of author's names, are numerous, contain the classical articles on a given subject as well as recent work. This is one of the outstanding features of this book.

To those who wish a brief but complete exposition of neurologic disorders of infants with adequate references, this work will prove valuable. The brevity with which many entities are treated is not a fault of the authors, but indicates how little is known of many fairly common disorders occurring in infancy.

The Child with Abdominal Pains

By John Apley, M.D., Consultant Pediatrician, United Bristol Hospitals and Bath Clinical Area, Shaw Lecturer in Diseases of Children, Bristol University. Springfield, Ill.: Charles C. Thomas, Publisher. Price $3.00.

This is a small book by an English pediatrician primarily interested in the preventive medicine, public health and statistical aspects of abdominal pain in children. The book is of only superficial clinical value. Especially emphasized are the psychic factors often operative in these problems. The author succeeds in placing the ubiquitous problem of the stomach-ache in the proper perspective in relation to the total gamut of childhood problems. The bibliography is selective but adequate for a work of this kind.

Cancer of the Breast

Edited by Willard H. Parsons, M.D. American Lecture Series. 226 pages. Springfield, Ill.: Charles C. Thomas, Publisher, 1959. Price $7.50.

This is another of the well-known American Lecture Series of books by Charles C. Thomas, Publisher. The monograph is a collection of chapters by well-known authorities in their field. An effort is made to cover the topic comprehensively, yet with an eye toward clinical applicability. Chapters are included on anatomy, surgical pathology, preoperative and postoperative care, the technic of simple, radical and superradical operation, and the role of x-ray and hormonal therapies.

The work is excellent for surgeons and oncologists but too detailed for those not especially interested in the subtleties of the subject.

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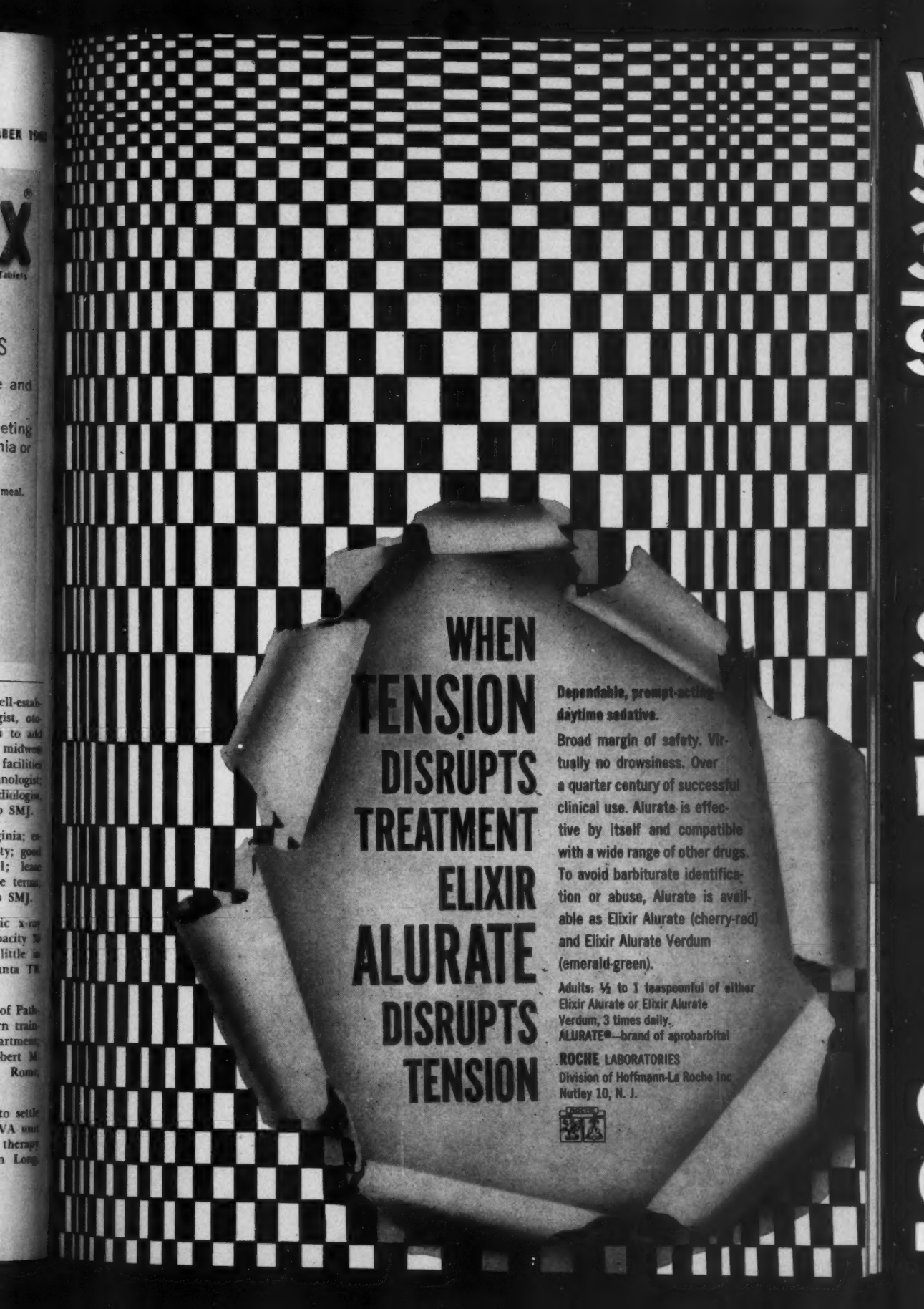
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Kestler, O.: Conservative Management of "Low Back Syndrome", J.A.M.A. 172: 2039 (April 30) 1960.

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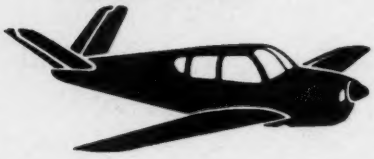

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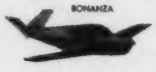

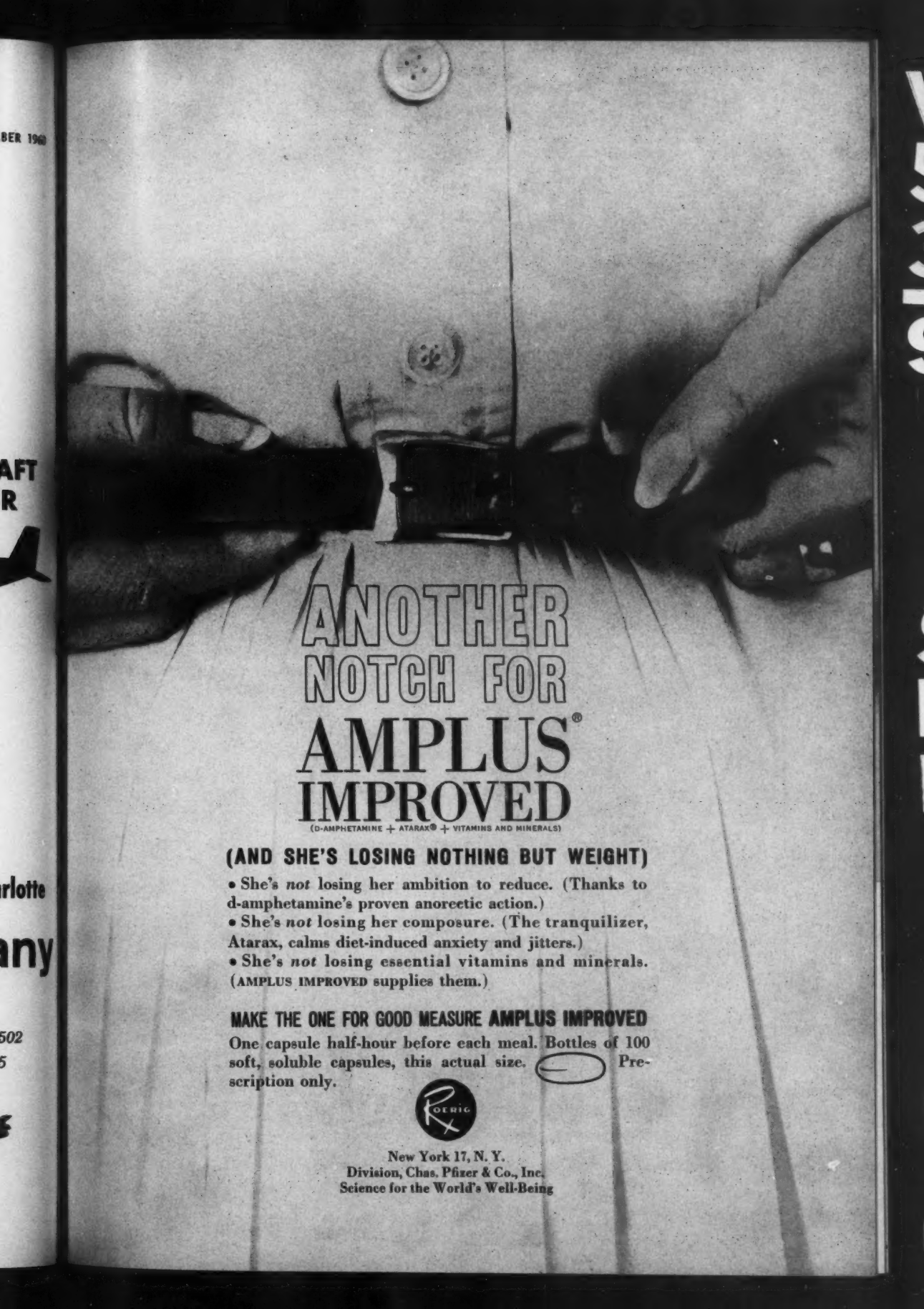
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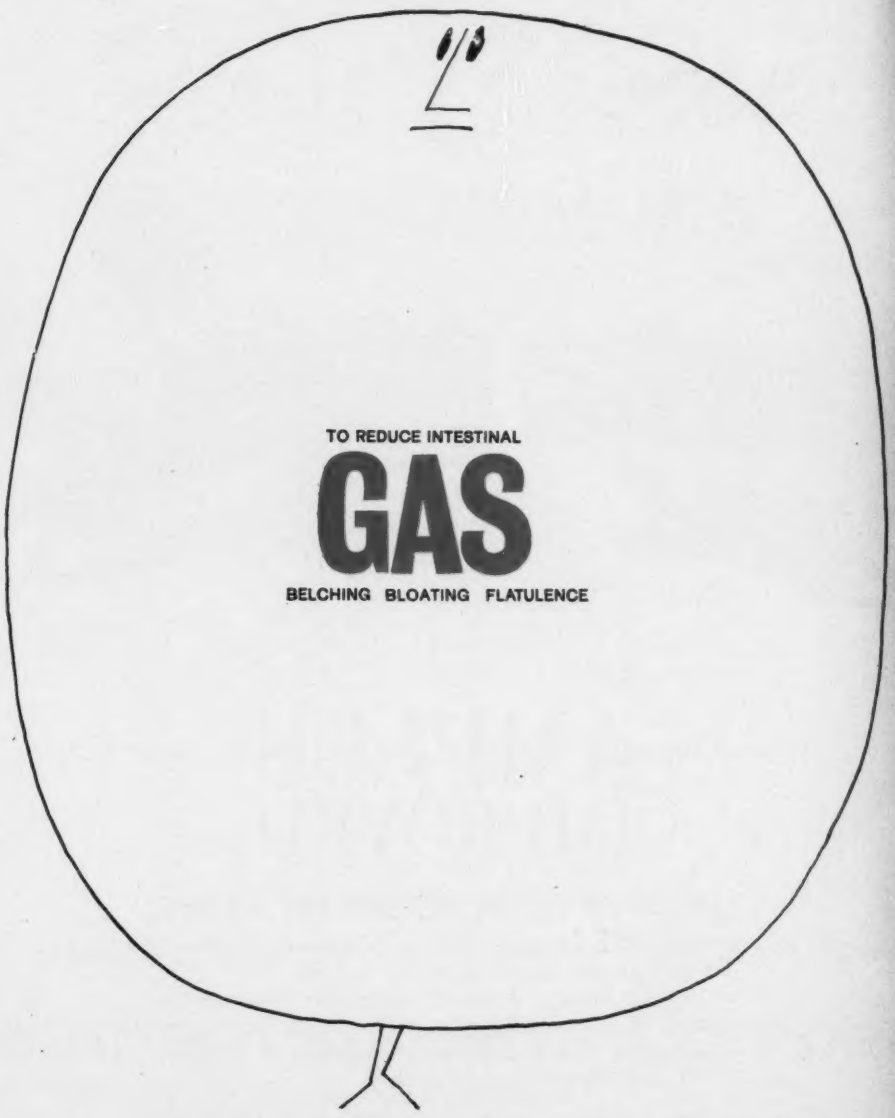

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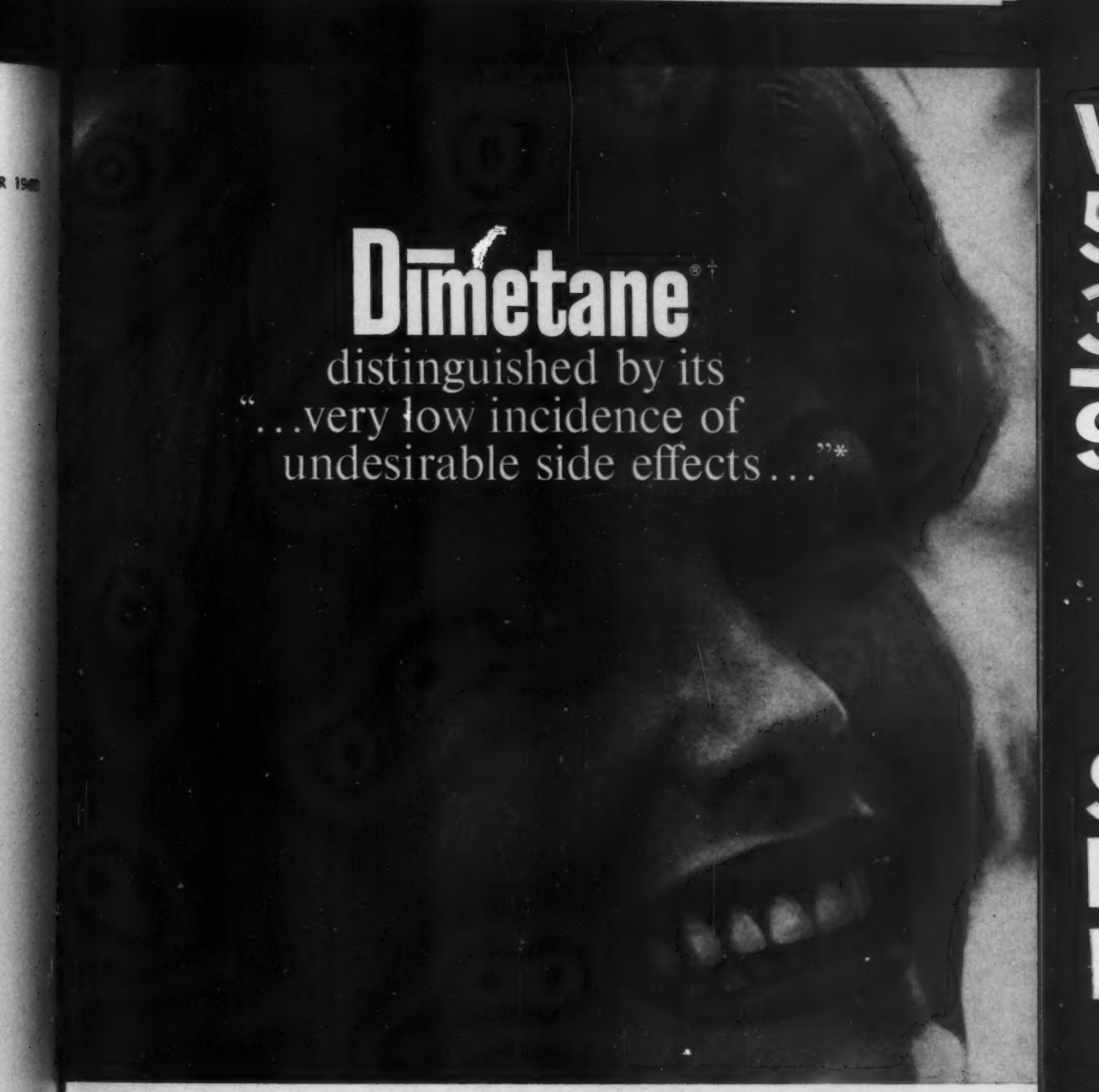

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Patients	200 infants and children, ages 2 months to 14 years
Diagnosis	Perennial allergic rhinitis
Therapy	Dimetane Elixir
Results	in 149, *good* results / in 40, *fair* results
Side Effects	Encountered in *only* 7 patients (in all except one, the side effect was mild drowsiness)

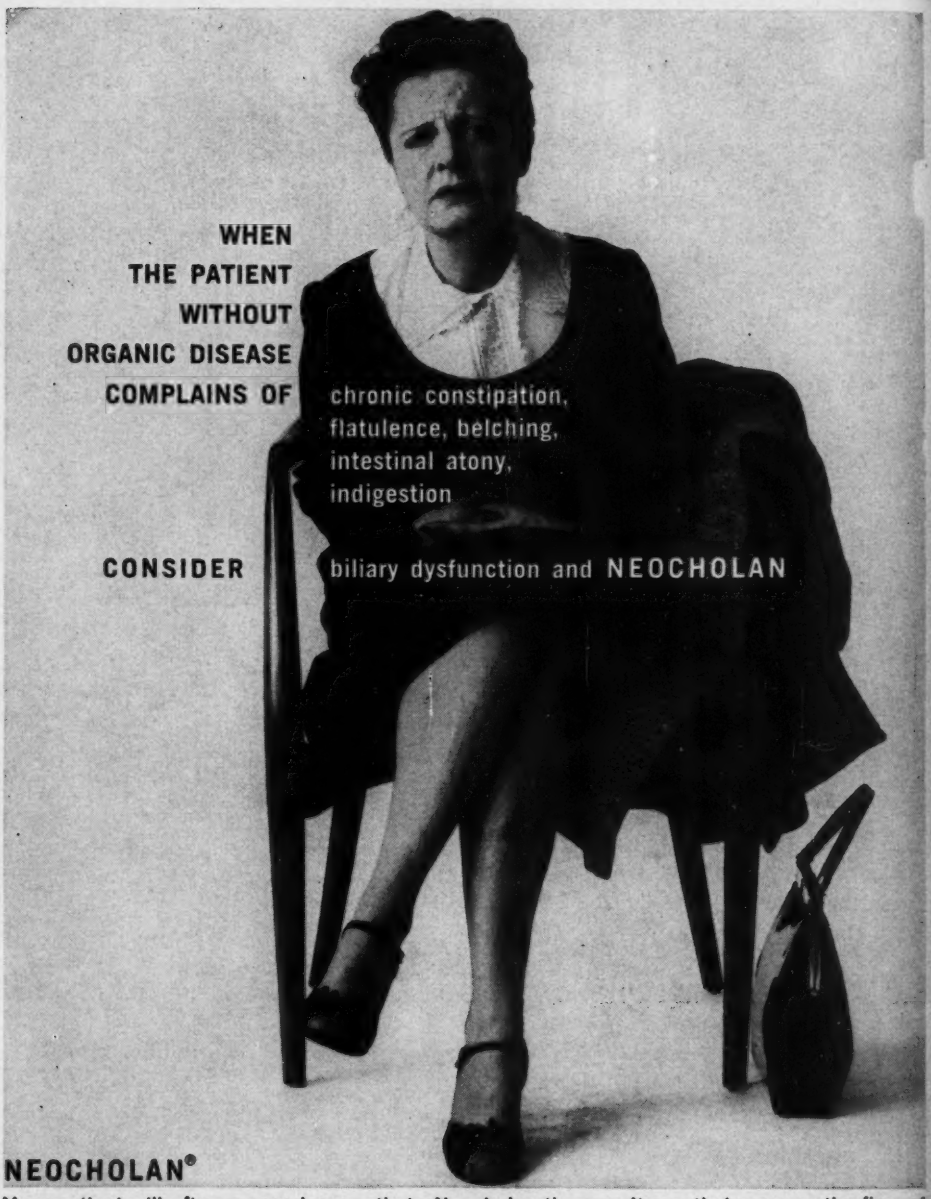
In allergic patients *of all ages,* Dimetane has been shown to work with an effectiveness rate of about 90% and to produce an exceptionally low incidence of side effects. Complete clinical data are available on request to the Medical Department. *Supplied:* DIMETANE Extentabs® (12 mg.), Tablets (4 mg.), Elixir (2 mg./5 cc.), new DIMETANE-TEN Injectable (10 mg./cc.) or new DIMETANE-100 Injectable (100 mg./cc.).

Robins

*MC GOVERN, J. P., MC ELHENNEY, T. R., HALL, T. R., AND BURDON, K. D.: ANNALS OF ALLERGY 17:925, 1959.

A. H. ROBINS CO., INC., RICHMOND 20, VIRGINIA / ETHICAL PHARMACEUTICALS OF MERIT SINCE 1878

† PARABROMDYLAMINE MALEATE



NEOCHOLAN®

Your patient will often respond promptly to Neocholan therapy. It greatly increases the flow of thin, nonviscid bile and corrects biliary stasis by flushing the biliary system. It also relaxes intestinal spasm, resulting in an unimpeded flow of bile and pancreatic juice into the small intestine. Neocholan helps to promote proper digestion and absorption of nutrients. It also encourages normal peristalsis by restoring intestinal tone.

Each tablet provides: Dehydrocholic Acid Compound, P-M Co. 265 mg. (Dehydrocholic Acid, 250 mg.); Homatropine methylbromide 1.2 mg.; Phenobarbital 8.0 mg. Supplied in bottles of 100 tablets.

PITMAN-MOORE COMPANY
DIVISION OF ALLIED LABORATORIES, INC.
INDIANAPOLIS, INDIANA

† PARABROMDYLAMINE MALEATE

Blood pressure that goes up with stress often comes down with SERPASIL®

(reserpine CIBA)

One reason that many cases of hypertension respond to Serpasil is that many cases are associated with stress. Stress situations produce stimuli which pass through the sympathetic nerves, constricting blood vessels, and increasing heart rate. Hyperactivity of the sympathetic nervous system may elevate blood pressure; if prolonged, this may produce frank hypertension. By blocking the flow of excessive stimuli to the sympathetic nervous system, Serpasil guards against stress-induced vasoconstriction, brings blood pressure down slowly and gently.

In mild to moderate hypertension, Serpasil is basic therapy, effective alone "...in about 70 per cent of cases..."*

In severe hypertension, Serpasil is valuable as a primer. By adjusting the patient to the physiologic setting of lower pressure, it smooths the way for more potent antihypertensives.

In all grades of hypertension, Serpasil may be used as a background agent. By permitting lower dosage of more potent antihypertensives, Serpasil minimizes the incidence and severity of their side effects.

CIBA
SUMMIT, N. J.

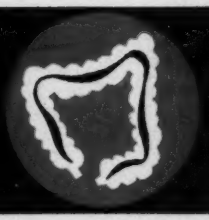
*Coan, J. P., McAlpine, J. C., and Boone, J. A.: J. South Carolina M. A. 51:417 (Dec.) 1955.

Complete information available on request.

Ulcerative Colitis - Still a Challenge!

Azulfidine®
BRAND OF SALICYLAZOSULFAPYRIDINE
-A Big Step Forward

For To-day's Patient - **A Brighter Outlook!**



Recent years have brought marked advances in the treatment of ulcerative colitis. Extensive clinical evidence supports the use of Azulfidine.

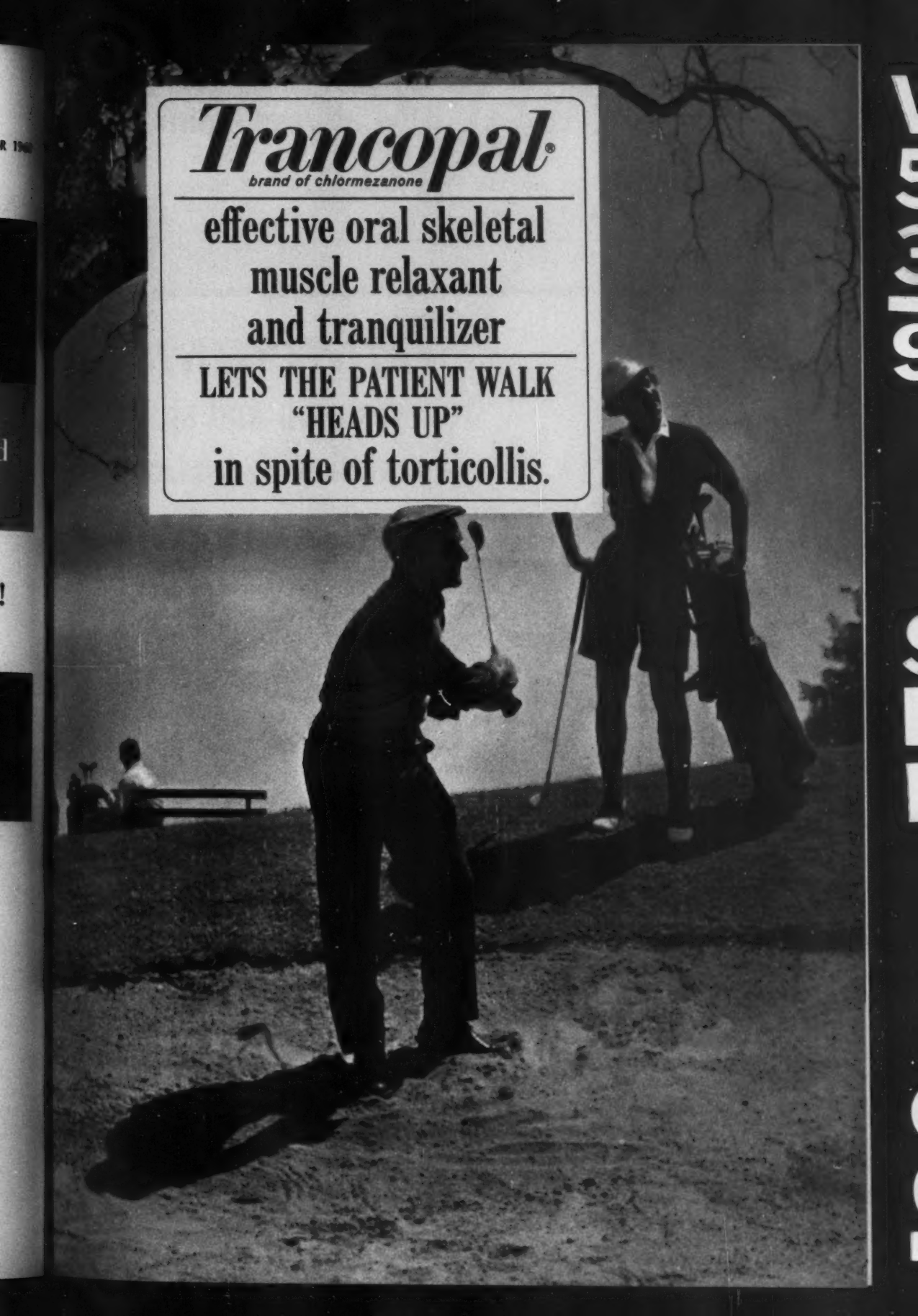
Azulfidine attacks the inflammatory and infectious phase of ulcerative colitis. Its effects are often striking, i. e.,

- *Bleeding and frequency of stools are sharply reduced.*
- *Abdominal cramping is controlled.*
- *Rectal mucosa in many cases heals within a month.*
- *Fever subsides, appetite improves.*

Treatment with Azulfidine means safe and reliable control of the distressing symptoms of ulcerative colitis.

Pharmacia Laboratories, Inc.
501 Fifth Avenue, New York 17, N. Y.

Film available on 'The Early Detection and Medical Management of Chronic Ulcerative Colitis' (16 mm., color, sound, showing time 31 minutes). Address requests to Ideal Pictures Inc., 58 E. South Water St., Chicago, Ill.


Trancopal®
brand of chlormezanone
effective oral skeletal
muscle relaxant
and tranquilizer
LETS THE PATIENT WALK
"HEADS UP"
in spite of torticollis.

Trancopal

relieves pain and spasm associated with torticollis.

In a recent study by Ganz, Trancopal brought considerable improvement or very effective relief to 20 of 29 patients with torticollis.[1] "The patients helped by the drug," states Ganz, "were able to carry the head in the normal position without pain." Similarly, Kearney found that in 8 of 13 patients with chronic torticollis treated with Trancopal improvement was excellent to good. "... Trancopal is the most effective oral skeletal muscle relaxant and mild tranquilizer currently available."[2]

Lichtman, in a study of patients with various musculoskeletal conditions, noted that 64 of 70 patients with torticollis obtained excellent to good relief with Trancopal.[3]

In a comparative study of four central nervous system relaxants, Lichtman reports that 26 of 40 patients found Trancopal to be the most effective drug.[3]

1. Ganz, S. E.: *J. Indiana* 52:1134, July, 1959. 2. Kearney *Current Therap. Res.* 2:117, 1960. 3. Lichtman, A. L.: *Acad. Gen. Pract. J.* 4:28, Oct.

Trancopal (brand of chlormezanone) and Caplets, trademarks reg. U. S. Pat. Off. 4716

Clinical results with *Trancopal*®[2]

	Excellent	Good	Fair	Poor	Total
W BACK SYNDROMES					
Acute low back strain	25	19	8	6	58
Chronic low back strain	11	5	1	1	18
"Porters' syndrome"*	21	5	1	1	28
Pelvic fractures	2	1	—	—	3
CK SYNDROMES					
Whiplash injuries	12	6	2	1	21
Torticollis, chronic	6	2	3	2	13
THER MUSCLE SPASM					
Spasm related to trauma	15	6	1	—	22
Rheumatoid arthritis	—	18	2	1	21
Bursitis	2	6	1	—	9
ENSION STATES	18	2	4	3	27
TOTALS	112 (51%)	70 (32%)	23 (10%)	15 (7%)	220 (100%

*Over-reaching in lifting heavy bags resulting in sprain of upper, middle, and lower back muscles.

Dosage: **Adults, 200 or 100 mg. orally three or four times daily.**
Relief of symptoms occurs in from fifteen to thirty minutes and lasts from four to six hours.

How Supplied: Trancopal Caplets®
200 mg. (green colored, scored), bottles of 100.
100 mg. (peach colored, scored), bottles of 100.

Winthrop LABORATORIES, New York 18, N. Y.

Trancopal

relieves pain and spasm associated with torticollis.

In a recent study by Ganz, Trancopal brought considerable improvement or very effective relief to 20 of 29 patients with torticollis.[1] "The patients helped by the drug," states Ganz, "were able to carry the head in the normal position without pain." Similarly, Kearney found that in 8 of 13 patients with chronic torticollis treated with Trancopal improvement was excellent to good. "... Trancopal is the most effective oral skeletal muscle relaxant and mild tranquilizer currently available."[2]

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Clinical results with *Trancopal*®[2]

	Excellent	Good	Fair	Poor	Total
LOW BACK SYNDROMES					
Acute low back strain	25	19	8	6	58
Chronic low back strain	11	5	1	1	18
"Porters' syndrome"*	21	5	1	1	28
Pelvic fractures	2	1	—	—	3
NECK SYNDROMES					
Whiplash injuries	12	6	2	1	21
Torticollis, chronic	6	2	3	2	13
OTHER MUSCLE SPASM					
Spasm related to trauma	15	6	1	—	22
Rheumatoid arthritis	—	18	2	1	21
Bursitis	2	6	1	—	9
TENSION STATES	18	2	4	3	27
TOTALS	112 (51%)	70 (32%)	23 (10%)	15 (7%)	220 (100%

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How Supplied: Trancopal Caplets®
200 mg. (green colored, scored), bottles of 100.
100 mg. (peach colored, scored), bottles of 100.

Winthrop LABORATORIES, New York 18, N. Y.

Continued from page 1197

Bill J. Swan, Concord; and Dr. Charles R. LaGrange, Bladenboro.

Dr. Donald M. Hayes has been appointed Assistant Dean of the Bowman Gray School of Medicine, it was announced recently by Dr. C. C. Carpenter, Dean of the Medical School. Dr. Hayes' appointment is effective immediately.

Three new faculty appointments have been announced for Bowman Gray School of Medicine: Dr. Henry S. Miller, Instructor in Internal Medicine; Dr. Herman E. Schmid, Jr., Instructor in Physiology and Pharmacology; and Dr. Robert P. Thomas, Instructor in Ophthalmology.

Dr. C. Hampton Mauzy, Professor of Obstetrics and Gynecology at Bowman Gray School of Medicine, has assumed supervision of obstetrics at the medical school and the North Carolina Baptist Hospital.

Dr. Ernest H. Wood, Professor of Radiology at the University of North Carolina School of Medicine, has been elected Vice-President of the American Board of Radiology.

The retiring Dean of the Duke University School of Medicine, Dr. W. C. Davidson, has been elected President of Alpha Omega Alpha, national medical honor society.

OKLAHOMA

Dr. Robert O. Ryan, Norman, has been elected Mayor of Norman by the City Commission.

The University of Oklahoma School of Medicine has announced several appointments and promotions. Two physicians have been added to the part-time faculty: Dr. Robert E. Ashley is Clinical Assistant in Psychiatry; and Dr. Louis J. Bernard is Clinical Assistant in Surgery. Moving up from Associate Professor to Professor are Dr. Alice M. Brues, in Anatomy; Dr. W. W. Schottstaedt, Chairman of the Department of Preventive Medicine and Public Health; and Dr. Ranwel Caputto, in Research Biochemistry. Those with the new title of Clinical Professor are Dr. Meredith M. Appleton and Dr. Jim M. Taylor, in Urology; Dr. Harry L. Deupree, in Obstetrics; Dr. Ella Mary George, in Physical Medicine; and Dr. Paul M. Vickers, in Surgery.

SOUTH CAROLINA

Dr. Ben N. Miller, Columbia, has been named President of the Duke Alumni Association.

Dr. Wightman R. Duke has joined the staff of St. Eugene Hospital in Dillon.

Colonel Roland K. Charles, Jr., has been appointed

Continued on page 68

NEW "TAILOR-MADE" ANTIHISTAMINE

TABLETS

keeps patient symptom-free, alert

Twiston* Rotoxamine

side effects, particularly drowsiness, negligible or absent

Twiston, 2 mg./Twiston R-A, 4 mg....

...full symptom-control with unusually low dosage...no toxicity report

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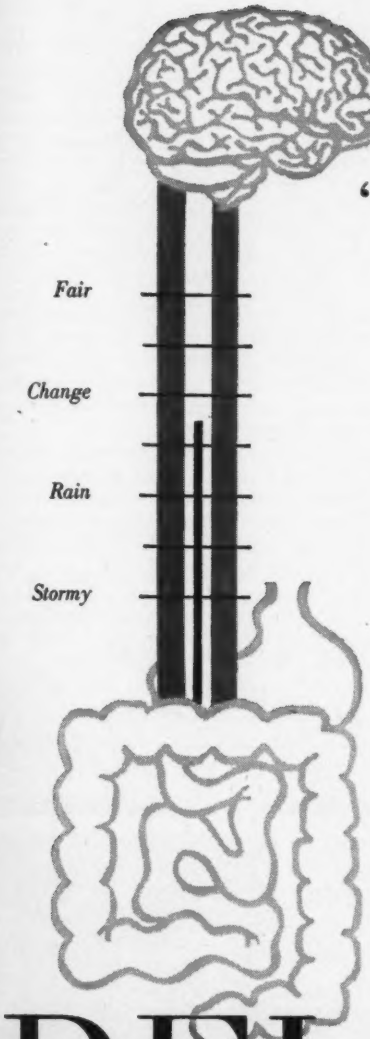

"the G-I tract is the barometer of the mind..."

Belbarb soothes the agitated mind and calms the G-I spasm through the central effect of phenobarbital and the synergistic action of fixed proportions of natural belladonna alkaloids on the gastrointestinal tract.

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SEDATIVE ANTISPASMODIC

20 years of clinical satisfaction

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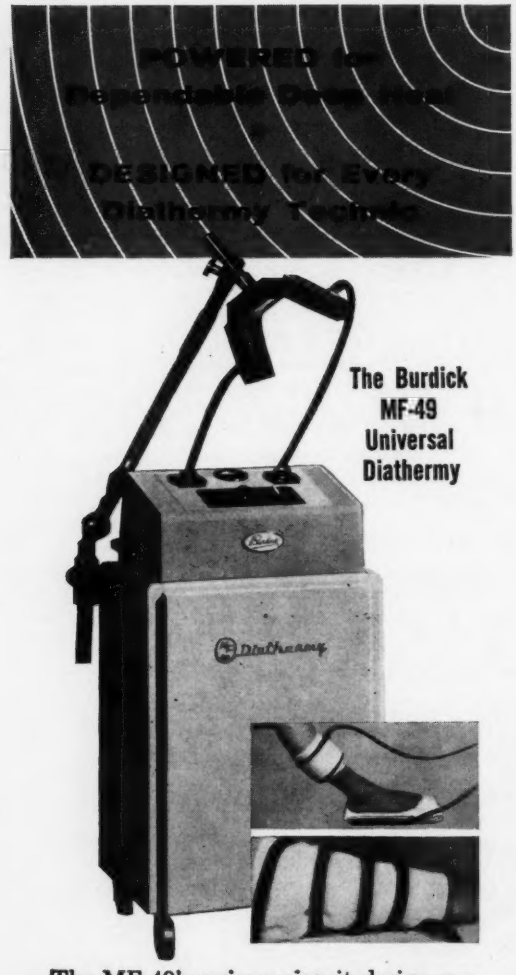

The MF-49's unique circuit design permits the use of full-power tube output for deep heating and treatment of large areas. Frequency is controlled by a separate tube circuit unaffected by the operating characteristics of the patient circuit. This results in high output relative to tube input.

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Continued from page 66

as Director of Medical Education for the Columbia Hospital of Richland County.

Officers of the South Carolina Pediatric Society include Dr. Kenneth H. Herbert, Charleston, President; Dr. Casper E. Wiggins, Greenwood, Vice-President; and Dr. Howard B. Smith, Conway, Secretary-Treasurer.

Dr. A. R. Johnston, St. George, is Second Vice-President of the Association of Surgeons of the Southern Railway System.

TENNESSEE

Dr. R. H. Kampmeier, Nashville, Editor of the *Southern Medical Journal*, has been installed as Chairman of the Board of Trustees of Senior Citizens, Inc.

Dr. Walter McLeod, Johnson City, is President of the Appalachian Chapter of the American Heart Association.

Officers of the West Tennessee Heart Association are Dr. John E. Neumann, Paris, President; and Dr. Laurence Jones, Union City, President-Elect.

Dr. Samuel S. Riven, Nashville, is President of the Tennessee Heart Association and Dr. Joseph E. Acker, Jr., Knoxville, is President-Elect.

Dr. Estle P. Muncy, Jefferson City, is the new President of the East Tennessee Heart Association, and Dr. James W. Richardson, Morristown, is on the Board of Directors.

Dr. Albert H. Fick, Humboldt, has been elected

Continued on page 80

NEO-VADRIN
ANTISEPTIC NASAL DECONGESTANT

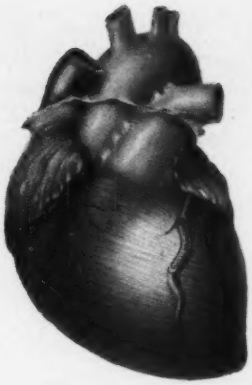
- Head Colds
- Hay Fever
- Sinusitis
- Rhinitis
- Allergy

The distinctive NEO-VADRIN formula is as follows:

dl-Norephedrine HCl	0.4%
Phenylephrine HCl	0.15%
Chlorobutanol	0.15%
Benzalkonium Chloride	0.005%

In 1 fluid ounce dropper bottles with special nasal applicator and ¾ ounce plastic spray bottles.

FIRST TEXAS *Pharmaceuticals, Inc.*
Pharmaceuticals Since 1901
DALLAS ATLANTA



IN ANGINA PECTORIS AND CORONARY INSUFFICIENCY

...the treatment must go further than vasodilation alone. It should also control the patient's ever-present anxiety about his condition, since anxiety itself may bring on further attacks.

AFTER MYOCARDIAL INFARCTION

...it is frequently not enough to boost blood flow through arterial offshoots and establish new circulation. The disabling fear and anxiety that invariably accompany the condition must be reduced, or the patient may become a chronic invalid.

Protects your coronary patient better than vasodilation alone

Unless the coronary patient's ever-present anxiety about his condition can be controlled, it can easily induce an anginal attack or, in cases of myocardial infarction, considerably delay recovery.

This is why Miltrate gives better protection for the heart than vasodilation alone in coronary insufficiency, angina pectoris and postmyocardial infarction. Miltrate contains not only PETN (pentaerythritol tetranitrate), acknowledged as basic therapy for long-acting vasodilation. What is more important — Miltrate provides Miltown, a tranquilizer of *proven* effectiveness in relieving anxieties, fear and day-to-day tension in over 600 clinical studies.

Thus, your patient's cardiac reserve is protected against his fear and concern about his condition...and his operative arteries are dilated to enhance myocardial blood supply.

Supplied: Bottles of 50 tablets. Each tablet contains 200 mg. Miltown and 10 mg. pentaerythritol tetranitrate.
Dosage: 1 or 2 tablets q.i.d. *before meals* and at bedtime, according to individual requirements.

REFERENCES

1. Ellis, L. B. *et al.*: Circulation *17*:945, May 1958. **2.** Friedlander, H. S.: Am. J. Cardiol. *1*:395, Mar. 1958. **3.** Riseman, J. E. F.: New England J. Med. *261*:1017, Nov. 12, 1959. **4.** Russek, H. I. *et al.*: Circulation *12*:169, Aug. 1955. **5.** Russek, H. I.: Am. J. Cardiol. *3*:547, April 1959. **6.** Tortora, A. R.: Delaware M. J. *30*:298, Oct. 1958. **7.** Waldman, S. and Pelner, L.: Am. Pract. & Digest Treat. *8*:1075, July 1957.

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Miltown® (meprobamate) + PETN

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Riker LABORATORIES, INC. NORTHRIDGE, CALIF.

Reduce frequency and severity of attacks

&

Lessen anxiety

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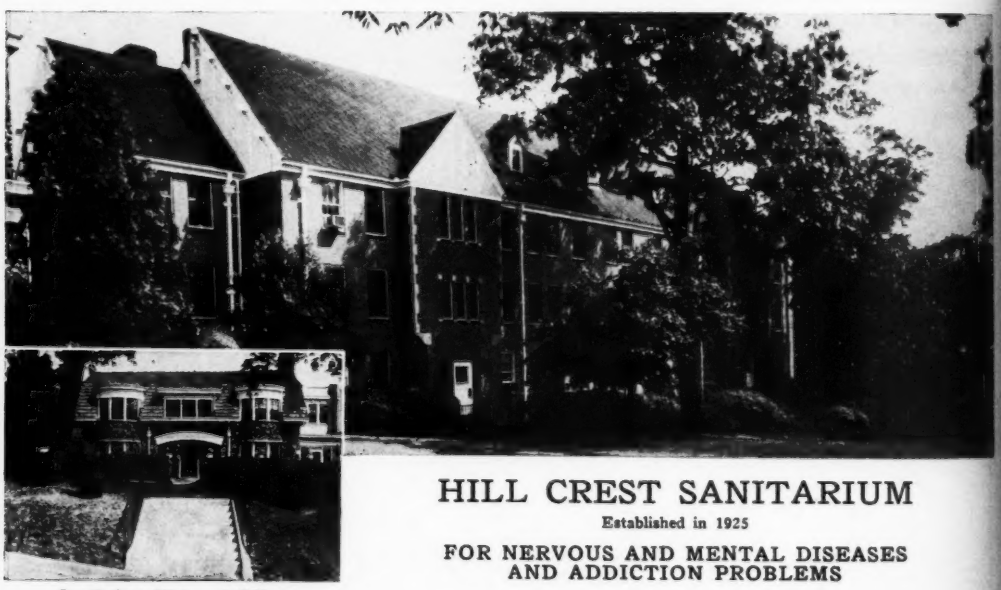
Tablets Containing Pentaerythritol Tetranitrate (PETN) 10 mg. and Rauwiloid® (Alseroxylon) 0.5 mg.



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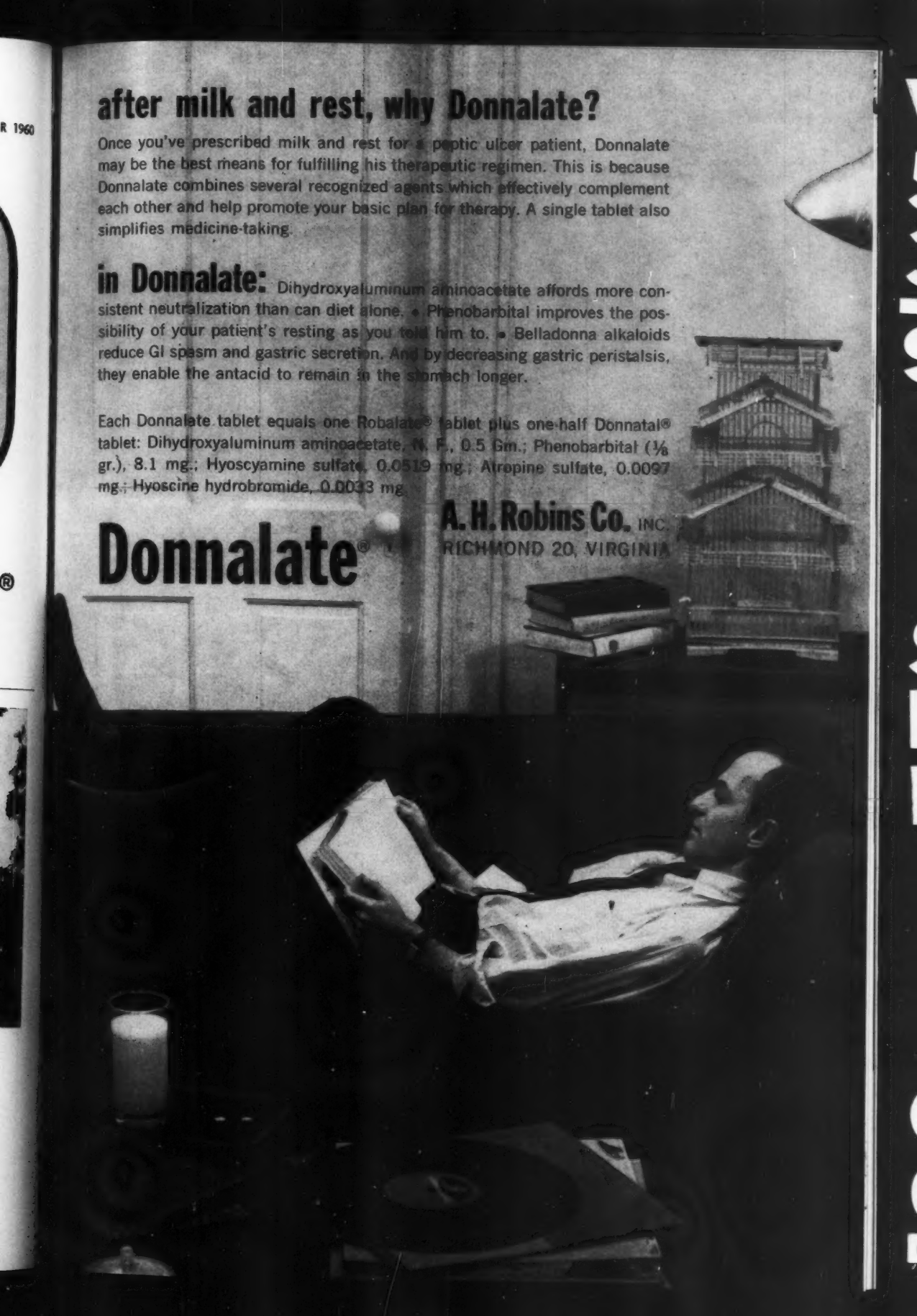
P. O. Box 2896, Woodlawn Station, Birmingham 6, Ala. Phone WO 1-1151 and WO 1-1152


after milk and rest, why Donnalate?
Once you've prescribed milk and rest for a peptic ulcer patient, Donnalate may be the best means for fulfilling his therapeutic regimen. This is because Donnalate combines several recognized agents which effectively complement each other and help promote your basic plan for therapy. A single tablet also simplifies medicine-taking.
in Donnalate: Dihydroxyaluminum aminoacetate affords more consistent neutralization than can diet alone. • Phenobarbital improves the possibility of your patient's resting as you told him to. • Belladonna alkaloids reduce GI spasm and gastric secretion. And by decreasing gastric peristalsis, they enable the antacid to remain in the stomach longer.
Each Donnalate tablet equals one Robalate® tablet plus one-half Donnatal® tablet: Dihydroxyaluminum aminoacetate, N. F., 0.5 Gm.; Phenobarbital (1/8 gr.), 8.1 mg.; Hyoscyamine sulfate, 0.0519 mg.; Atropine sulfate, 0.0097 mg.; Hyoscine hydrobromide, 0.0033 mg.
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A. H. Robins Co. INC.
RICHMOND 20, VIRGINIA

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take a breather pardner...

Little cold sufferers take to Rhinall Nose Drops without a fuss! Pleasant, fast-acting, easy to use... and so economical!

Relieves nasal congestion in
colds
sinusitis
allergic rhinitis
no burning or irritation
no after reactions
no risk of sensitization

Contains:
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in an isotonic saline menstruum

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A LOGICAL ADJUNCT TO THE WEIGHT-REDUCING REGIMEN

meprobamate **plus** d-amphetamine... reduces appetite...elevates mood...eases tensions of dieting...**without** overstimulation, insomnia or barbiturate hangover.

Dosage: One tablet one-half to one hour before each meal.

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meprobamate 400 mg., with d-amphetamine sulfate 5 mg., Tablets

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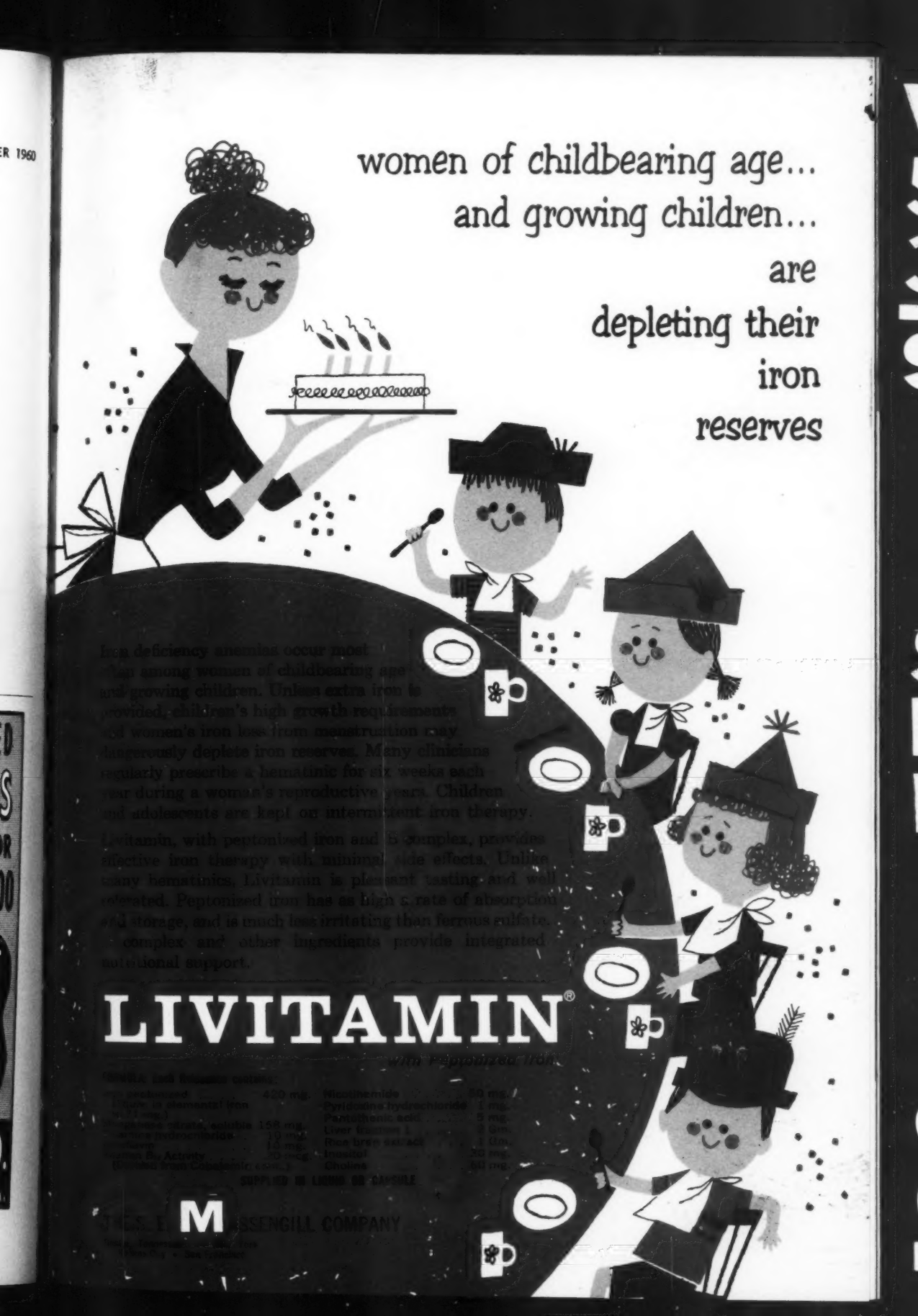
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and growing children...
are
depleting their
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reserves
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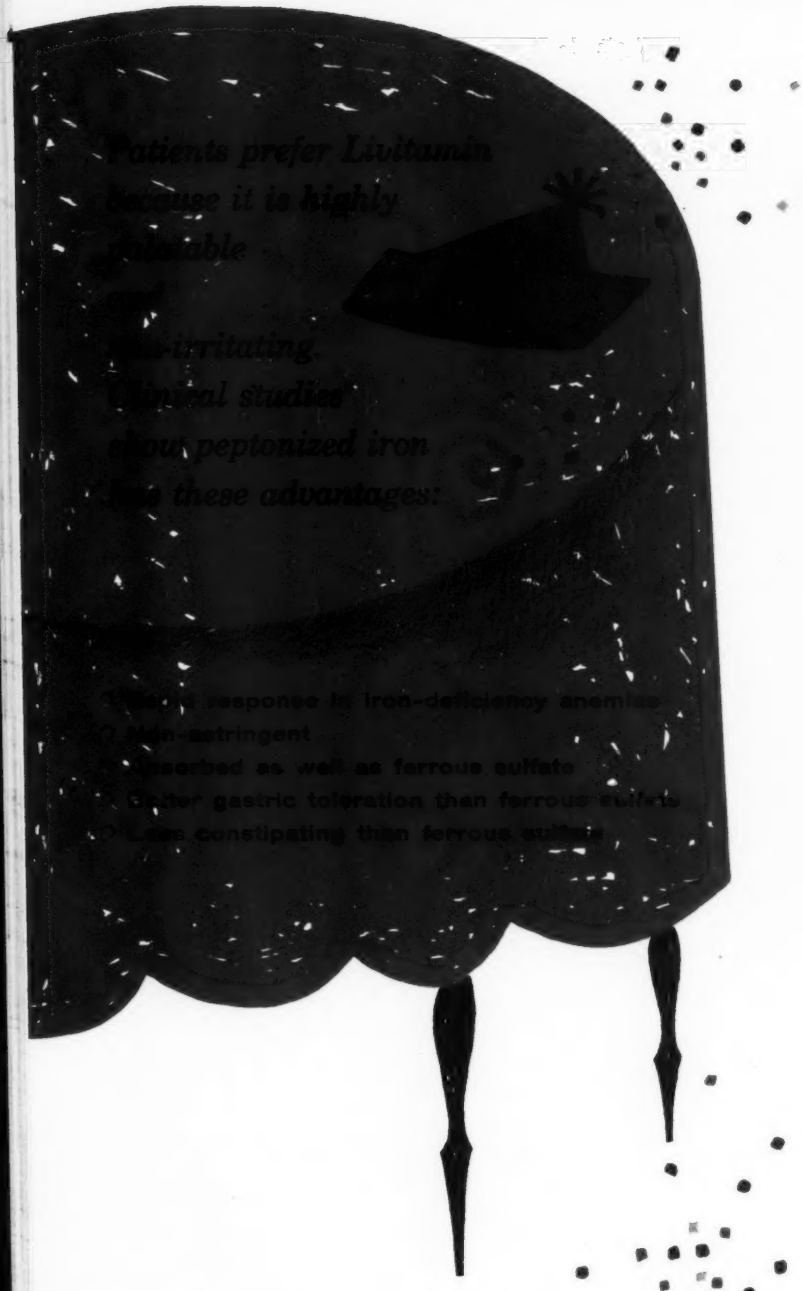

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•Keith, J.H.: Utilization and Toxicity of Peptonized Iron and Ferrous Sulfate, Am. J. Clin. Nutrition 1:35 (Jan.-Feb., 1957).

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Cremomycin® provides rapid relief of virtually all diarrheas

NEOMYCIN—rapidly bactericidal against most intestinal pathogens, but relatively ineffective against certain diarrhea-causing organisms.

SULFASUXIDINE® (succinylsulfathiazole)—an ideal adjunct to neomycin because it is highly effective against Clostridia and certain other neomycin-resistant organisms.

KAOLIN AND PECTIN—coat and soothe the inflamed mucosa, adsorb toxins, help reduce intestinal hypermotility, help provide rapid symptomatic relief.

For additional information, write Professional Services, Merck Sharp & Dohme, West Point, Pa.

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CREMOMYCIN AND SULFASUXIDINE ARE TRADEMARKS OF MERCK & CO., INC.

Cartoon idea by pharmacist Emil Magdalener

Many of you may have seen a recent cartoon depicting a midnight scene in front of a pharmacy. A woman is pounding on the door and the pharmacist is leaning out the window of his apartment over the store. "Open up," shouts the woman. "My husband is sick and I need a stamp so I can send this prescription to the mail order house."

The drug that always fails is the drug that isn't there

Far-fetched? Perhaps, but there are those who would have us believe that our present system of drug distribution is inefficient and costly, and should be replaced by presumably more efficient and cheaper centralized or bureaucratic methods. Disregarding the probable political philosophy behind these suggestions, consider what a marvelously intricate and efficient system of drug distribution we have in this country. • From the laboratories of the manufacturers comes a steady stream of new and better drugs for your patients. Warehoused and stocked by drug wholesalers, these products are available in over 53,000 pharmacies scattered across the length and breadth of our land. And woe to the pharmacist who hasn't been provided with yesterday's laboratory discovery for your use in treating a patient today. • The economists speak of "utility of time" and "utility of place." We simply say that you can confidently prescribe *what* you choose, *when* it is needed, *wherever* your patient may be.

This message is brought to you by the producers of prescription drugs as a service to the medical profession. For additional information, please write Pharmaceutical Manufacturers Association, 1411 K Street, N.W., Washington 5, D.C.

this hypertensive patient prefers Singoserp... and so does his physician

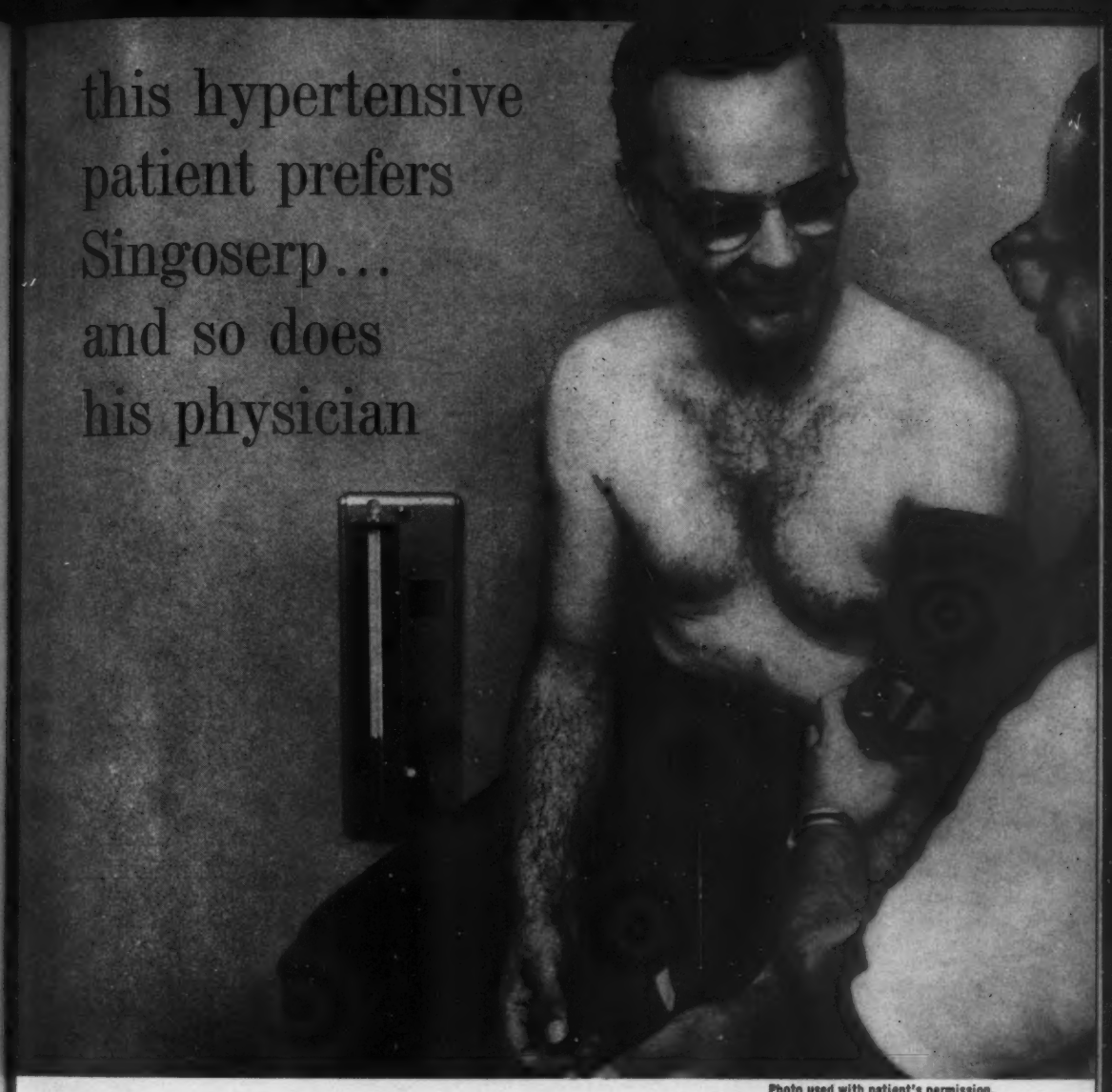

Photo used with patient's permission.

Patient's comment: "The other drug [whole root rauwolfia] made me feel lazy. I just didn't feel in the mood to make my calls. My nose used to get stuffed up, too. This new pill [Singoserp] doesn't give me any trouble at all."

Clinician's report: J. M., a salesman, had a 16-year history of hypertension. Blood pressure at first examination was 190/100 mm. Hg. Whole root rauwolfia lowered pressure to 140/80 — but side effects were intolerable. Singoserp, 0.5 mg. daily, further reduced pressure to 130/80 and eliminated all drug symptoms.

Many hypertensive patients and their physicians prefer Singoserp® because it usually lowers blood pressure without rauwolfia side effects

SUPPLIED: Singoserp **Tablets**, 1 mg. (white, scored). Also available: Singoserp®-Esidrix® **Tablets** #2 (white), each containing 1 mg. Singoserp and 25 mg. Esidrix; Singoserp®-Esidrix® **Tablets** #1 (white), each containing 0.5 mg. Singoserp and 25 mg. Esidrix. **Complete information sent on request.**
Singoserp® (syrosingopine CIBA) Singoserp®-Esidrix® (syrosingopine and hydrochlorothiazide CIBA)

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BAMADEX®

meprobamate 400 mg., with d-amphetamine sulfate 5 mg., Tablets

meprobamate **plus** d-amphetamine...
depresses appetite...elevates mood...
eases tensions of dieting...**without** over-stimulation, insomnia or barbiturate hangover.

Dosage: One tablet one-half to one hour before each meal.

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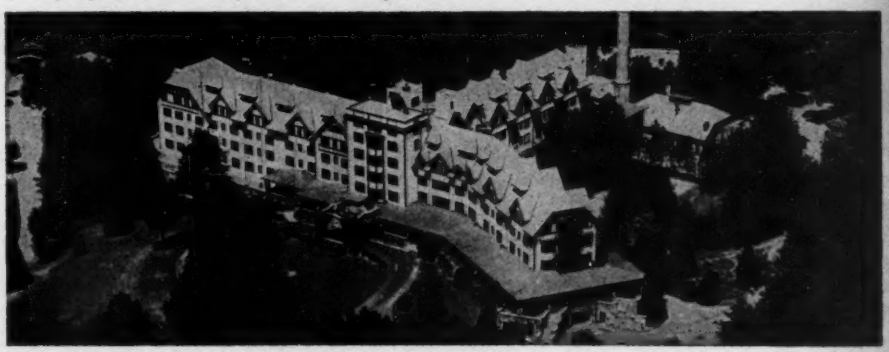

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References: 1. David, N. A.; Porter, G. A., and Gray, R. H.: Monographs on Therapy 5:60 (Feb.) 1960. 2. Friend, D. H.; Clin. Pharm. & Therap. 1:6 (Mar.-Apr.) 1960. 3. Ford, R. V.: Current Therap. Res. 2:92 (Mar.) 1960.

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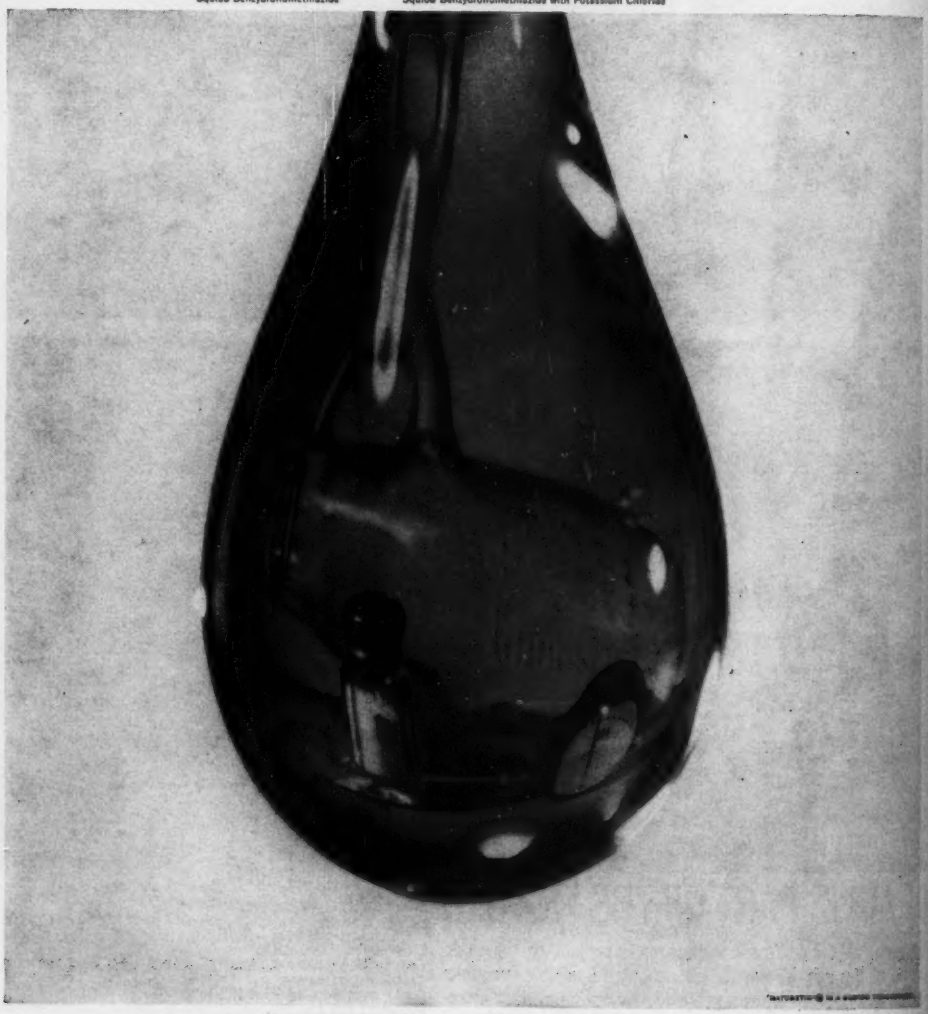

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Continued from page 68

President of the medical staff of St. Mary's Hospital. Other officers are Dr. E. C. Crafton, Vice-President; and Dr. James W. Hail, Secretary, both of Trenton.

President of the Tennessee Pediatrics Society is Dr. Felix G. Line, Knoxville.

Dr. Samuel L. Raines, Memphis, has been re-elected Secretary of the American Urological Association.

Officers of the West Tennessee Medical and Surgical Association include Dr. W. A. Anderson, Dyersburg, President; Dr. Leland Johnston, Jackson, First Vice-President; and Dr. James Carroll, Savannah, Second Vice-President.

Dr. E. P. Mobley, Jr., has been elected Chief of Staff of the Henry County General Hospital. Dr. Kenneth Ross is Vice-Chief and Dr. Ike Jones is Secretary. All of these physicians are from Paris.

Officers of the staff of the Blount Memorial Hospital include Dr. William C. Crowder, Chief; Dr. G. T. Proctor, Vice-Chief; and Dr. Cecil B. Howard, Secretary. All are from Maryville.

Dr. Hollis E. Johnson, Nashville, is President-Elect of the American College of Chest Physicians.

TEXAS

The University of Texas Postgraduate School of Medicine and the University of Texas M. D. Anderson Hospital and Tumor Institute will co-sponsor a Clinical Symposium on Pediatric Oncology in Houston, November 11 and 12, 1960. The program will include lectures by guest speakers and by members of the M. D. Anderson Hospital staff, as well as case presentations and panel discussions. Important aspects of chemotherapy, surgery and radiotherapy in the management of children with various types of neoplastic disease will be covered. For further information write to the Office of the Dean, University of Texas Postgraduate School of Medicine, 410 Jesse Jones Library Building, Texas Medical Center, Houston 25, Texas.

Officers of the Texas Diabetes Association are Dr. James A. Greene, Houston, President; Dr. Harold L. Dobson, Houston, President-Elect; Dr. J. Wilson David, Corsicana, First Vice-President; Dr. Warren W. Moorman, Fort Worth, Second Vice-President; and Dr. John W. Chriss, Corpus Christi, Secretary-Treasurer.

Dr. Gray Carter, Dallas, is President of the Texas Society of Gastroenterologists and Proctologists. Other officers are Dr. Francis M. Spencer, San Angelo, First Vice-President; Dr. Robert J. Rowe, Dallas, Second Vice-President; and Dr. A. C. Broders, Jr., Temple, Secretary-Treasurer.

The Texas Society of Plastic Surgeons announces its officers: Dr. John B. Patterson, Fort Worth; Dr. Robert J. Wise, Houston, Vice-President; and Dr. Raymond O. Brauer, Houston, Secretary-Treasurer.

Officers of the Texas Association of Public Health Physicians are Dr. Luther P. Walter, Austin, President; Dr. B. M. Primer, Austin, President-Elect; Dr. Albert G. Randall, Amarillo, Vice-President.

Dr. Isaac S. McReynolds, Houston, has been elected President of the Texas Orthopedic Association; Dr.

Continued on page 82

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Continued from page 80

Herbert E. Hipps, Waco, is Vice-President; and Dr. Margaret Watkins, Dallas, is Secretary-Treasurer.

The Texas Industrial Medical Association has elected Dr. Noble B. Daniel, Texarkana, President; Dr. Robert A. Wise, Houston, President-Elect; Dr. Carl A. Nau, Galveston, Vice-President; and Dr. J. G. Burdick, Pasadena, Secretary-Treasurer.

Officers of the Texas Traumatic Surgical Society include Dr. Edward R. Rowe, Galveston, President; Dr. Frank Kidd, Dallas, First Vice-President; Dr. C. M. Ashmore, Houston, Second Vice-President; and Dr. John C. Long, Plainview, Secretary-Treasurer.

Dr. Otto Lippman, Austin, is President of the Texas Ophthalmological Association. Other officers include Dr. Jack B. Lee, San Antonio, Vice-President; Dr. James H. Scruggs, Waco, Secretary; and Dr. John C. Kuppinger, Harlingen, Treasurer.

The Texas Air-Medics Association has elected the following officers: Dr. C. D. Henry, San Antonio, President; Dr. Virgil Payne, Dallas, President-Elect and Vice-President; and Dr. C. F. Miller, Waco, Secretary-Treasurer.

Elected to office for the Texas Society of Athletic Team Physicians are Dr. William S. Horn, Jr., Fort Worth, President; Dr. Jack G. Brannon, Houston, President-Elect; Dr. James R. Whitehurst, Houston, Vice-President and Program Chairman; and Dr. Louis J. Levy, Fort Worth, Secretary-Treasurer.

Officers of the Texas Neuropsychiatric Association are Dr. Clarence S. Hoekstra, Dallas, President; Dr Robert L. Stubblefield, Dallas, President-Elect; Dr. M. Lake Fowler, Jr., Vice-President; and Dr. E. Ivan Bruce, Jr., Galveston, Secretary-Treasurer.

The Texas Otolaryngological Association has chosen the following officers: Dr. August J. Streit, Amarillo, President; Dr. Jack Lee Turner, Odessa, President-Elect; Dr. William Skokan, Fort Worth, Vice-President; Dr. Louis E. Adin, Jr., Dallas, Secretary; and Dr. Dor W. Brown, Jr., Fredericksburg, Treasurer.

Dr. Hiram M. Anderson, San Angelo, is President of the Texas Chapter, American College of Chest Physicians. Other officers are Dr. Carlos J. Quintanilla, Harlingen, First Vice-President; Dr. John W. Middleton, Galveston, Second Vice-President; and Dr. Milton V. Davis, Dallas, Secretary-Treasurer.

The Texas Society of Anesthesiologists announces its officers: Dr. Randle J. Brady, Houston, President; Dr. Merle D. Thomas, El Paso, President-Elect; Dr. M. T. Jenkins, Dallas, Vice-President; and Dr. Eugene L. Slataper, Houston, Secretary.

Dr. Malcolm W. Harrison, Houston, is President of the Texas Dermatological Society; Dr. J. Fred Mullins, Galveston, Vice-President; Dr. D. Shelton Blair, Dallas, Secretary-Treasurer; and Dr. William F. Spiller, Jr., Houston, Program Chairman.

Dr. E. Richard Halden, Fort Worth, is Vice-President of the South Central Association of Blood Banks.

Dr. G. W. N. Eggers, Galveston, has been elected President of the American Board of Orthopedic Surgeons.

Continued on page 86

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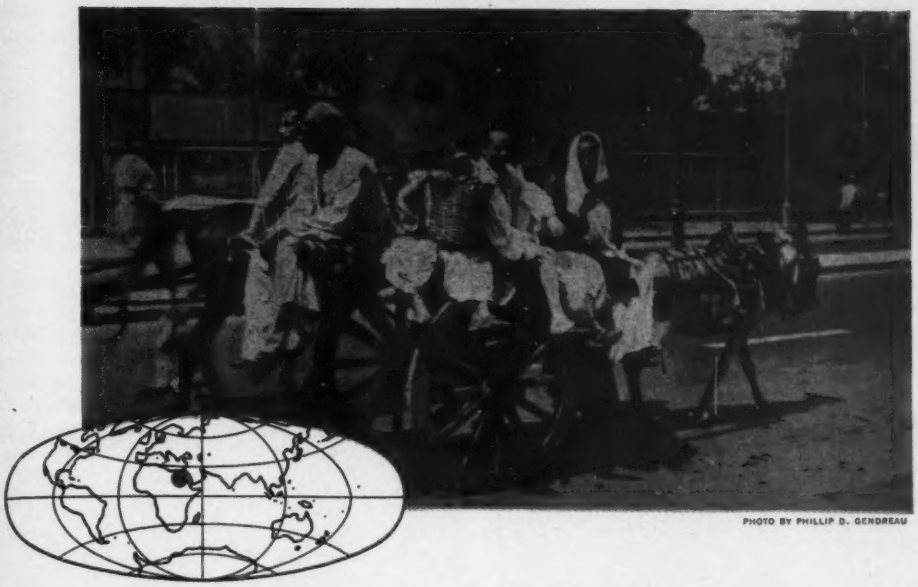

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Musgrave, M. E. and Arm, H. G.: Antibiotic Med. & Clin. Therapy 7:17 (Jan.) 1960.

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Continued from page 82

Dr. J. Layton Cochran, San Antonio, has been chosen a regional director of the American Cancer Society.

VIRGINIA

Officers of the Virginia Academy of General Practice include Dr. Boyd H. Payne, Staunton, President; Dr. William J. Hagood, Jr., Clover, President-Elect; Dr. A. L. VanName, Jr., Urbanna, Vice-President; and Dr. S. F. Driver, Roanoke, Secretary.

Dr. Robert H. Harrington, Marion, has been elevated to the Eminent Grand Commandership of the Knights Templar in Virginia.

Dr. Lionel M. Lieberman, Hampton, is Chairman of the Hampton Jewish Community Council.

New members of The Medical Society of Virginia are Dr. Jose D. Coll, Richmond; Dr. Robert W. Fry, Suffolk; Dr. Pierce D. Nelson, Norton; Dr. Robert J. O'Donnell, Falls Church; Dr. Walter S. Price, Hampton; Dr. Lily Ruckstuhl, Fairfax; Dr. Stanley M. Sager, Winchester; Dr. David H. Smith, Fairfax; and Dr. Anthony V. Torre, Roanoke.

WEST VIRGINIA

Dr. Albert C. Esposito, Huntington, presented a paper on "Cataract and Allied Ocular Surgery" at the Section of Ophthalmology of the International College of Surgeons in Rome, Italy, during May. Dr. Esposito is Secretary of the Section on Ophthalmology and Otolaryngology for Southern Medical Association.

The Annual Meeting of the West Virginia Heart Association is scheduled for Sept. 16, 1960, at the Chancellor Hotel in Parkersburg. The subject for the meeting is "Cardiac and Industry." For further information write Dr. Frederick S. Kelley, Executive Director, West Virginia Heart Association, 1906 Washington Street East, Charleston.

Dr. Frank V. Langfitt, Clarksburg, has been given the honorary degree of Doctor of Humanities by Salem College.

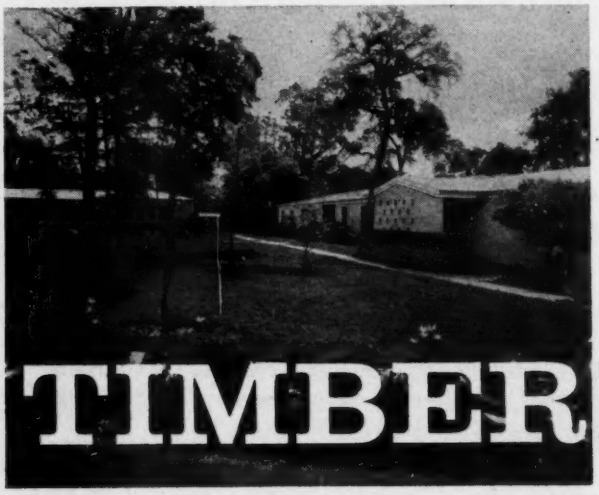
Dr. Emma Jane Freeman, Buckhannon, has been appointed Director of the Division of Maternal and Child Health of the State Department of Health.

Dr. Charles A. Hoffman, Huntington, has been named a member of the Professional Relations Committee of the national Blue Shield Medical Care Plans.

Dr. Raphel J. Condry, Elkins, has accepted appointment as Chief of Medical Service at the Veterans Administration Hospital in Clarksburg.

Dr. William L. Cooke, Charleston, has been elected a member of the Board of Directors of the Alumni Association of the Medical College of Virginia.

New members of the West Virginia State Medical Association are Dr. Benjamin H. Biddle, Nutter Fort; Dr. John J. Bryan, Bluefield; and Dr. Edward A. Gretchen, Weirton.



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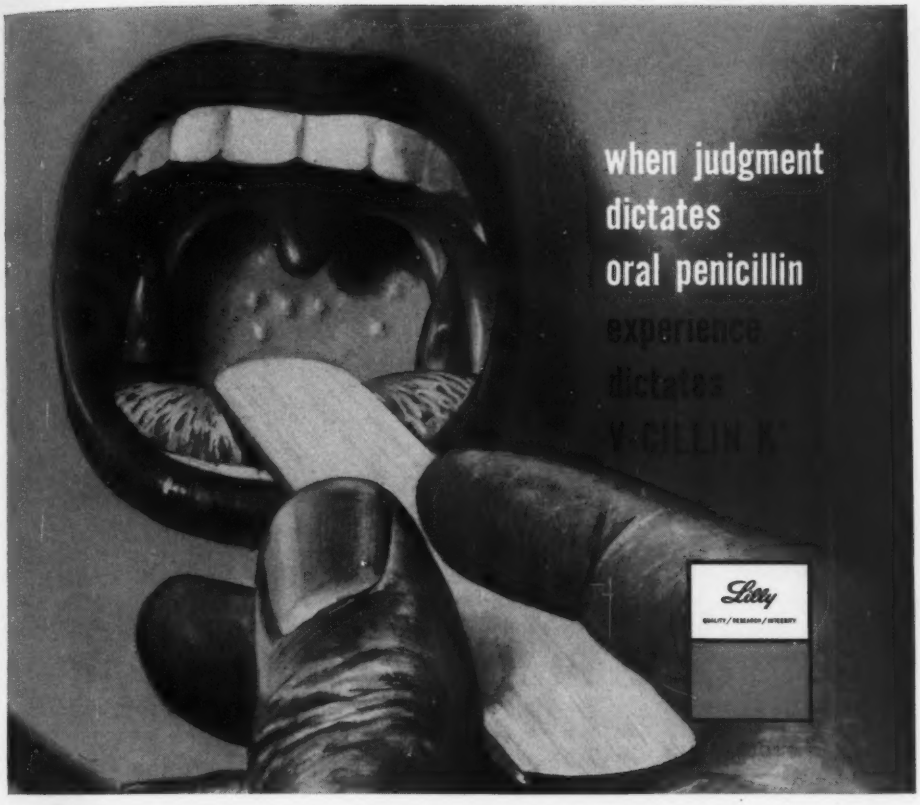

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1. Griffith, R. S.: Comparison of Antibiotic Activity in Sera Following the Administration of Three Different Penicillins, Antibiotic Med. & Clin. Therapy, 7: No. 2 (February), 1960.

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